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Quinidine Therapy in the Treatment of Cardiac Irregularities Due to Hyperthyroidism*†

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QUINIDINE sulphate has rightfully earned a permanent place in the treatment of cardiac irregularities, especially those which are secondary to hyperthyroidism. The heart rhythm naturally tends to return to normal after thyroidectomy alone but by the addition of quinidine therapy the number of restorations to normal rhythm can be increased greatly.

The percentage of cases in which the heart rhythm returns to normal after thyroidectomy alone depends chiefly on the following factors: (1) the duration of the irregularity of the heart rhythm previous to operation; (2) the degree of arteriosclerosis and myocardial degeneration; (3) the age of the patient; (4) complications, such as focal infections.

In 1927, I reported a series of 75 cases of hyperthyroidism in nearly all of which auricular fibrillation had been present for a long time, and in several cases extensive cardiac failure had been present for several years. Our follow-up records showed that in only 32 per cent of these cases did the rhythm return to normal. Later in the same year I reported another series of 75 cases which included all patients who had shown any auricular fibrillation while in the hospital. Sixty per cent of this second series acquired normal rhythm. Quinidine was used three times. In a later series of cases the normal rhythm was restored in 66 per cent.

At that time quinidine was not being used directly after operation, and the difficulty of instituting treatment a few weeks later led to the omission of its use in a large number of cases. Also, it was found that our attempts to restore normal rhythm a few weeks or months after thyroidectomy frequently met with failure. This fact led to the institution of quinidine therapy while the patient was still in the hospital. At first it was administered on the sixth or seventh postoperative day, and no embolic accidents resulted from its use. However, by that time the patient was out of bed and ready to go home and did not wish to remain in the hospital for two or three days longer in order to undergo treatment for the heart.

We then tried instituting quinidine therapy during the decline of the postoperative reaction, that is, on the third or fourth postoperative day. The good results of this experiment would seem to indicate that this is the best time to

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institute treatment as the patient is still in bed and his stay in the hospital need not be prolonged after he is able to be up.

No attempt is made to restore the heart to a normal rhythm before operation because it is seldom successful, and if it does succeed, auricular fibrillation is likely to recur at the time of the operation. For this reason quinidine is seldom used after a first lobectomy.

At the Cleveland Clinic we have adopted the following plan of management of patients with hyperthyroidism in whom abnormal heart rhythm is present:

All patients in whom auricular fibrillation is still present on the third postoperative day are reported. A test dose of quinidine is administered to these patients and if no ill effects are noted they receive 5 grs. of quinidine sulphate every four hours, day and night, for twenty-four hours. The pulse is counted before the administration of each dose and if the rhythm is found to be regular, medication is discontinued. If no improvement is noted after twenty-four hours treatment, 5 grs. of quinidine are given every three hours for twenty-four hours and then every two hours for twenty-four hours, and occasionally for forty-eight hours.

In the majority of cases normal rhythm will be restored after a few doses, but occasionally two or three days' treatment will be necessary. In one case we found no change after the third day, and treatment was continued on the fourth day. Two hours after the last dose of quinidine had been administered, normal rhythm was restored.

In the case of one of our patients, a woman 46 years of age, a very interesting result from the use of quinidine was noted. Symptoms of hyperthyroidism had been present for two years previous to thyroidectomy. The thyroid tissue showed diffuse hyperplasia. After thyroidectomy had been performed, the general condition improved greatly but the patient was nervous and her heart continued to fibrillate. The administration of digitalis did not slow the ventricular rate to normal although it reduced it from 130 to 100. The patient's basal metabolic rate was +23, suggesting that residual hyperthyroidism was present. The administration of iodine had no effect on the heart action. Inasmuch as there was no recurrent enlargement of the thyroid gland, quinidine therapy was instituted with the result that the heart rhythm became normal, the nervousness disappeared, and the patient was able to return to work. Since that time, a year ago, there has been no recurrence of symptoms.

A very few patients are sensitive to quinidine. In one of our recent cases, the patient experienced a great deal of distress during one night after her third dose of 5 grs. of quinidine. She became dyspneic, nauseated, flushed, perspired a great deal, and experienced fear of death. It can not be stated definitely whether this condition was due to the effect of the quinidine or to a pulmonary embolus but the fibrillation persisted, and the patient refused further treatment.

In the case of another patient quinidine therapy had to be discontinued on account of nausea and vomiting. Its administration was attempted a second time with a similar result.

The question is frequently asked—will auricular fibrillation recur after the heart rhythm has been made regular by the administration of quinidine? We know of only one case in which there was a recurrence of auricular fibrillation after a thyroidectomy had restored the heart rhythm to normal, in death. For several days the temperature varied between 103° and 105.5° and the pulse rate varied between 130 and 160, but at no time was any auricular fibrillation present.

In table I is shown the effect of thyroidectomy on auricular fibrillation.

Table II shows the results of the use

TABLE I

	Regular after thyroidectomy	Irregular after thyroidectomy
Series I	24	51
75	32%	68%
Series II	45	30
75	60%	40%
Series III	112	73
185	60.5%	39.5%
Series IV	26	31
57	45.7%	54.3%

TABLE II

Treated with quinidine	Regular after quinidine	Irregular after quinidine
late	10	6
16	60%	40%
prompt	22	1
23	96%	4%

and in the group in which quinidine therapy was instituted there has not been any recurrence of auricular fibrillation.

In one case, a patient was given quinidine on the fourth postoperative day and the heart rhythm became regular. The following day auricular fibrillation was again present. Upon inquiry it was found that only a lobectomy had been performed. It is my opinion that if auricular fibrillation recurs after quinidine has been administered postoperatively, residual or recurrent hyperthyroidism may always be suspected.

In another case the patient was given quinidine about six months after a thyroidectomy had been performed. After the third day of treatment the heart rhythm was found to be restored to normal. A few months later an abdominal operation was performed, followed by a general peritonitis resulting of quinidine in a series of cases with persistent auricular fibrillation after thyroidectomy. In sixteen cases quinidine therapy was instituted during a period of from several weeks to several months following operation, and of this group the normal heart rhythm was restored in 60 per cent of the cases while in 40 per cent the rhythm remained irregular. In 1930 a follow-up record was made of 57 cases following thyroidectomy. Quinidine therapy was instituted promptly in the treatment of 23 patients resulting in the restoration of the normal heart rhythm in 96 per cent.

When auricular fibrillation is still present after the fourth postoperative day, it usually persists unless quinidine therapy is instituted, and our experience has demonstrated that better results are obtained if quinidine is given during the decline of the postoperative reaction.

Auricular Flutter

In my experience the complication of auricular flutter associated with hyperthyroidism has been encountered only three times, and in two of these cases auricular fibrillation and auricular flutter were present alternately. Of the three patients who were treated with quinidine, normal heart rhythm was restored in two cases but not in the other, although a very thorough course of quinidine therapy was given. Auricular fibrillation was present in the latter case and after the administration of quinidine the heart rhythm became regular but the pulse rate remained at about 100. This condition was unusual, as the pulse rate after the heart rhythm has been restored to normal is usually about 80. An electrocardiogram showed a flutter which later was unaffected by four days' treatment with quinidine

Conclusions

1. Quinidine sulphate is a very useful drug for the restoration of a normal cardiac rhythm if a thyroidectomy alone fails to achieve this result.

2. Observations over several years show that the maximum benefit is obtained when the drug is given on the third or fourth postoperative day.

3. A very few patients are sensitive to quinidine, thus prohibiting its use in such cases.

4. By adding quinidine therapy after thyroidectomy when auricular fibrillation is still present, a normal cardiac rhythm will be restored in about 96 per cent of cases.

Tachycardia*†

By Chas. W. Barrier, M.D., *Fort Worth, Texas*

To a noted cardiologist of the previous generation, paroxysmal tachycardia was merely a clinical curiosity. To the few who have attacks prolonged into weeks and even years and who suffer acute heart failure when an extra burden such as a cold and cough is put upon them, it is a tragedy.

I am limiting my subject to auricular and nodal tachycardia. The literature does not indicate the number subject to these attacks. This paper is based upon a study of twenty-six proven cases occurring in about four thousand general admissions in three years. If to this twenty-six are added those who gave histories of paroxysmal acceleration, but who were without electrocardiograms, the incidence of paroxysmal tachycardia would be higher than is generally thought.

Etiology

Experimentally, Lewis[1] has produced attacks by stimulation of the vagus nerve and by ligating the coronary artery.[2] Boorman[4] destroyed the sinus node with radon, but this was often eventually followed by a rhythm simulating a typical sinus rhythm. Bouchut[3] reports a case with an infarct in the region of the sinus node. Clinically, Major[5] found acute and chronic myocarditis in the auricle of a person dying in an attack, and Anderson[6] reports auricular tachycardia as a complication of diphtheria. Attacks are not unusual in cases of mitral stenosis, and they are likely to occur in the course of heart failure from various causes. Kern[7] reports a case occurring on inflation of the Fallopian tubes with air. It is interesting to conjecture that the attack was due to air embolism of the coronary artery, basing our conjecture upon the frequency of tachycardia after occlusion and of the occurrence of coronary embolism when air enters the circulation.

An important exciting cause of tachycardia is drugs, chief among which is digitalis, as pointed out by Luten.[8] Howard[9] calls our attention to the great danger of excessive digitalis producing a coexistent auricular and ventricular tachycardia. Galli,[10] contrary to the experience of Lewis, insists that atropine can excite paroxysms. Adrenalin will frequently cause attacks in susceptible patients. Terrell[11] has observed nodal attacks after ephedrine in an asthmatic who had not previously had attacks. Numerous other drugs, as well as mineral salts, may provoke

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them. In spite of the efficacy of quinidine in stopping attacks, Korns[12] labels it as an excitant of tachycardia.

The great majority suffering from tachycardia are free of cardiac pathology. Carr[13] stresses its occurrence among the nervous type, and Thomas[14] reports cases occurring in persons subject to other neuroses such as migraine.

Two of my cases had early exophthalmic goiter, one had rheumatic endocarditis, a few had mild hypertension, and several fell in the sclerotic heart group; but most were without heart lesions. Two were conspicuous because of their athletic prowess. Yet, even these had marked nervous instability, one of them blushing, trembling and turning giddy when called on in class.

Colgate[15] reports cases occurring in early infancy, and while cases occur at any age, the literature would indicate the prevalence of tachycardia in the fifth and sixth decade. My cases fall roughly into two age groups; the first is that of adolescence in which the individuals have excessive variability of their sinus rate and an increased emotivity suggesting in many a degree of hyperthyroidism. The other group consists mostly of women about the menopause who are often overworked, poorly nourished, suffering from the effects of purgatives and flatulence, and frequently from migraine attacks. In both, attacks are precipitated most often by unusual exertion or emotions.

Mechanism

Lewis[16] teaches that the mechanism of an attack is repeated ectopic systoles, a different mechanism from flutter which is a circus movement. Colgate[15] observed cases in which it was difficult to tell the difference in electrocardiograms between cases of tachycardia and flutter. He concluded that the mechanism of tachycardia was a circus movement varying only in degree from that of flutter. One would conclude from Crawford's[17] case in which there was a shift of the pacemaker from the sinus to the lowest part of the A-V node that the attacks are neurogenic. Willius dismisses the idea that toxins make these hearts unusually susceptible to nervous influences. The view of Otto,[18] arrived at from the action of drugs on the onset and arrest of attacks, that the mechanism is a circus movement, seems the most plausible.

Prognosis

To Willius [19] the prognosis depends upon the underlying heart pathology and upon the rate and duration of the attacks. Many texts speak of the prolonged attacks ending in congestive heart failure and death. Carr[13] believes that death is a rare termination. One of my patients who had mitral stenosis and a crippled myocardium entered the hospital on the thirteenth day of an attack, cyanotic, with a bloody sputum and general anasarca. Recovery took place. Another who endured attacks lasting for months with a rate of about 160 without trouble except for a limitation of his efforts, developed acute congestive heart failure after a cold and cough, and death was imminent for days. The usual rate of 160 increased to 200 and the mechanism from auricular to nodal.

Prognosis, however, does not concern itself so much with mortality as with incapacity. This incapacity in short attacks varies from concern over

the rate and unpleasant palpitation to more serious symptoms such as cardiac pain,[20] syncope and convulsions;[21] but as a rule the general comfort and lack of dyspnea are striking when the excessive rates are considered. Nodal tachycardia causes more symptoms than an auricular attack of the same rate due, no doubt, to poorer filling of the ventricle and the contraction of the auricle against a closed valve. One patient with a mild exophthalmic goiter, B.M.R. +35, experienced no real inconvenience from a sinus rate of 150, but complained of severe heart pains, violent palpitation, nausea, vomiting, and fainting at a sudden onset of nodal tachycardia with a rate of only 160.

In some patients, prolonged duration is the chief disturbing factor. Most authors report the time as only a few minutes and at the longest as only a matter of weeks. Cohn speaks of attacks extending into years, and Gilbert[22] reports a case which he considered permanent. One of my cases lasted seven days, one thirteen days, one six weeks, one three years save for one thirty minute interval, and one case for the past six years has been in permanent tachycardia except when under the effects of digitalis. The case lasting three years had for years been subject to frequent short attacks in which there was fainting, but after the onset of permanent tachycardia the rate dropped to between 160 and 180 and she lived in comparative comfort. The other case in which the tachycardia was permanent was comfortable while at quiet occupations, but on strenuous exercise the rate would rise to above 200 with distressing dyspnea.

These patients with prolonged attacks, unless victims of obvious heart lesions, have suffered no cardiac deterioration from the rate.

Diagnosis

The characteristics of paroxysmal auricular and nodal tachycardia upon which a diagnosis is made clinically are the abrupt onset and arrest, repetition of attacks, constancy of the rate throughout an attack, the much quoted observation of Feil[24] of the absolute regularity of rhythm, and the effect of vagus stimulation in arresting an attack.

It is not unusual, however, for auricular attacks to begin at rates above 180 and finally drop to 140 or less. The rate may increase because of exercise or atropine, and the rhythm may be irregular due to vagus effect or drugs, digitalis in one case producing a bigeminy; and a fast sinus rate may unnoticeably pass into an auricular tachycardia of the same rate by a gradual shift of the pacemaker.

Treatment

Treatment consists in removing the cause, arresting and preventing attacks. Two of our cases were permanently cured by iodine and thyroidectomy. Most attacks are short and end spontaneously. Often rest, chloral and bromides are necessary. All stimulants such as camphor should be prohibited.

Vagal stimulation has been the most popular mode of arrest. Fiessinger[25] has obtained this end by depriving the patient of air causing forced respiration. Wolffe[26] stops attacks by intravenous calcium, the action being very similar to quinidine; while Steppe[27]

has made use of the vagotonic action of cholin. Wilson[28] publishes cases showing arrest by intravenous digitalis. Eakin[29] would use digitalis with caution in tachycardia unless the attack is known to be supraventricular because of its danger in ventricular tachycardia, especially in the presence of coronary occlusion.

Bodin[30] first used quinidine, showing that it would stop some attacks, while only slowing the rate in others; and Iliescu[31] publishes a case in which the rate was slowed to the normal rate without a change of mechanism. Sprague[32] believes half the cases can be arrested by quinidine and shows its value in preventing attacks.

We have treated eight patients in repeated attacks with digitalis, either with massive doses by mouth or intravenously. Arrest was obtained in six cases, and was felt in all probability to be due to the digitalis. In ten cases we have used quinidine sulphate either by mouth or vein and arrested attacks in nine. There were five of these cases that responded both to digitalis and quinidine. Two patients, one because of apparently permanent tachycardia or frequent attacks, were rationed on quinidine for a year. Attacks were certainly warded off, but the dose had to be increased from a start of six grains a day to twenty-one. Even at this dose attacks appeared and the patients complained so vehemently of the drug that it was withdrawn. These two cases and one other case of permanent tachycardia were kept on digitalis for the greater part of two years. Though large doses, four to six grains a day, were often required, the results on the whole were more satisfactory.

Mode of Action of Drugs

As the mode of action of drugs in stopping attacks is of supreme interest, careful records of the action of digitalis and quinidine given intravenously were made in the following cases:

Case I. A woman of thirty-nine, whose examination, made later, revealed no finding varying from normal except a blood pressure of 148, entered the hospital one and one-half hours after the onset of an attack, in circulatory collapse. She had a nodal tachycardia, rate 255. Three hours after the onset 8 cc. of digalen were given by vein, and in seven minutes the rate was slowed to 220. After a second dose of 6 cc., sinus mechanism was restored, rate 90. (See figure 1.)

Case II. A vigorous man of thirty-two without any heart lesion came to the Clinic because of attacks of tachycardia which had come at frequent intervals since age sixteen. They were usually ushered in by exertion or emotion and lasted from one to fourteen hours. It always required a period of rest before cessation of an attack.

On the second morning an attack of nodal tachycardia, rate 260, was induced by ten minutes of vigorous exercise. This attack was allowed to subside spontaneously. After about half an hour the rate had slowed gradually to 200. In another hour the rate suddenly dropped from 200 to 90. During the next twenty minutes there was frequent repetition of the fast rhythm, but finally the slow rate was established.

On the next morning another attack was induced. In one minute after the injection of only 0.05 gram of quinidine sulphate the rate had appreciably slowed. A tracing one minute after an injection of 0.1 gram of quinidine sulphate showed a sinus rhythm with a rate of 150, which in four minutes had dropped to 110. The total dose was 0.25 gram, and the total time thirty-four minutes. (See figure 2.) The mode of arrest was quite a contrast to the spontaneous one. The total dose might have been given at one time and no doubt arrest would have been sudden.

A third attack was induced the next morning. In one minute after 5 cc. of digifolin, the rate had dropped from 255 to 240 and at the end of seven minutes to 195, and at the end of thirty minutes after a total dose of 20 cc. the rate was 180 and markedly irregular. Three minutes later normal rhythm ensued before inversion of the T-wave. (See figure 3.)

Case III. A woman of twenty-one without a heart lesion came for examination in 1928 saying that five years before she had had her first attack of fast heart. These attacks became more frequent and extended, so that for the last three years she had been in a constant attack. Nodal tachycardia, rate 160, was found. Quinidine sulphate caused a normal rhythm of 100. As this gave so much relief she was kept on quinidine for the year of 1928. At first six grains a day were sufficient, but by the end of the year twenty-one grains were necessary and attacks became so frequent and the effects of the large doses so annoying that quinidine was discontinued.

It was then found that digitalis in large doses would ward off attacks, and for 1929 and part of 1930 she stayed on digitalis. While attacks occurred she felt that her condition was distinctly better than when on no treatment and on quinidine, but in June, 1930, she stopped digitalis and found that she was free of attacks unless she lost sleep or overworked. When attacks occurred, they were more violent and had to be stopped with quinidine.

Five hours after the onset of an attack on January 18, 1931, she entered the hospital before any treatment. Nodal tachycardia,

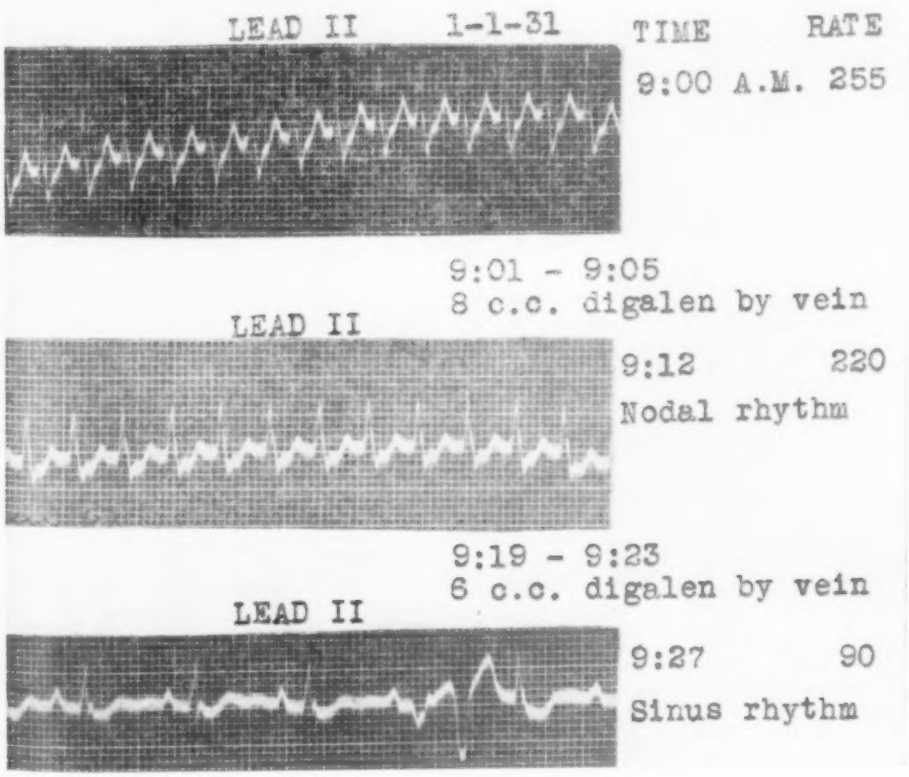

FIG. 1. Nodal tachycardia. Arrested with digitalis.

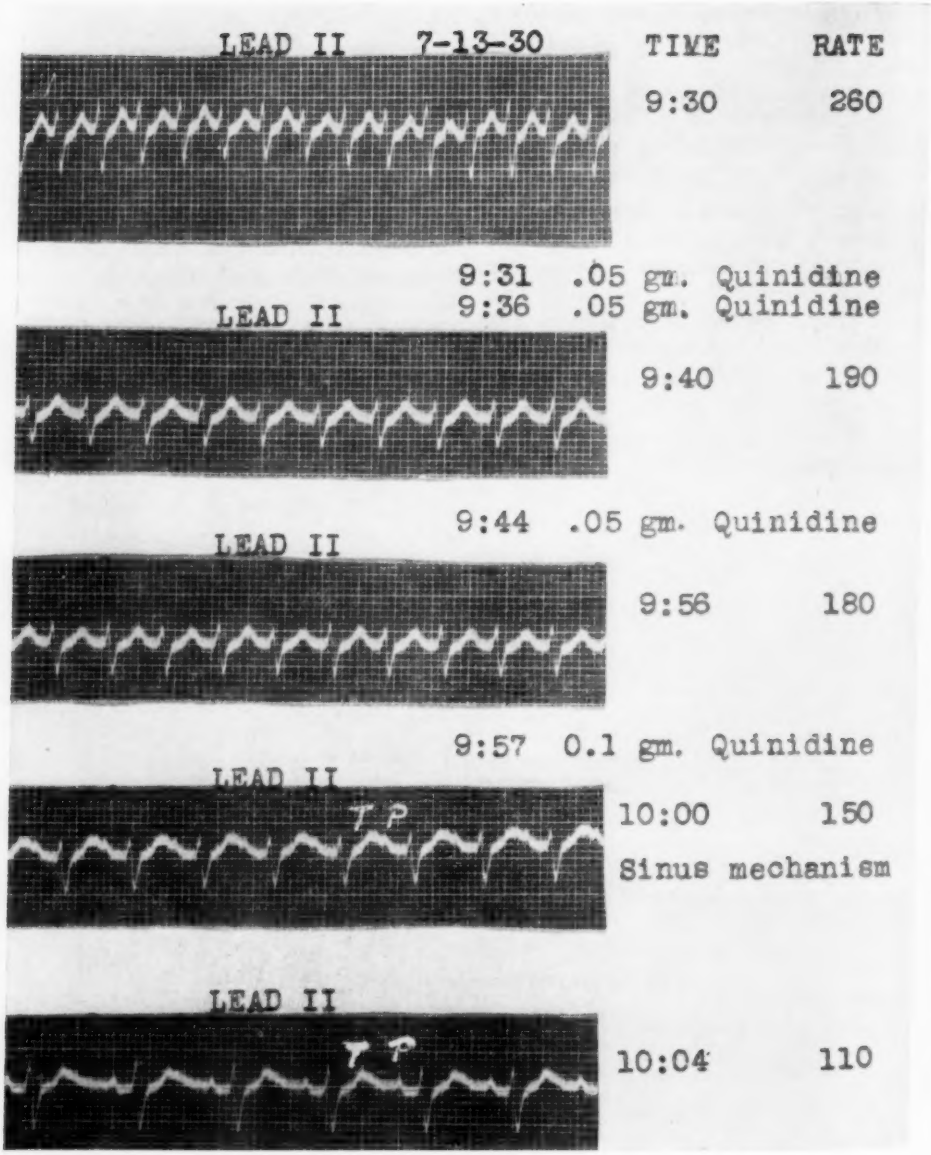

FIG. 2. Nodal tachycardia. Tracing every three minutes; string under constant vision.

rate 195, was present. Three minutes after the injection of 0.2 gram of quinidine, normal rhythm resulted, rate 145. In forty minutes this had gradually slowed to 110. (See figure 4.)

Nine days later she again entered the hospital, twelve hours after the onset of

Case IV. A boy of twenty-one, robust in every respect and without heart lesion, first came under observation four years ago. His physician had records showing a tachycardia for the previous two years. His was an auricular tachycardia, rate 160 to 190. Repeated efforts with quinidine failed

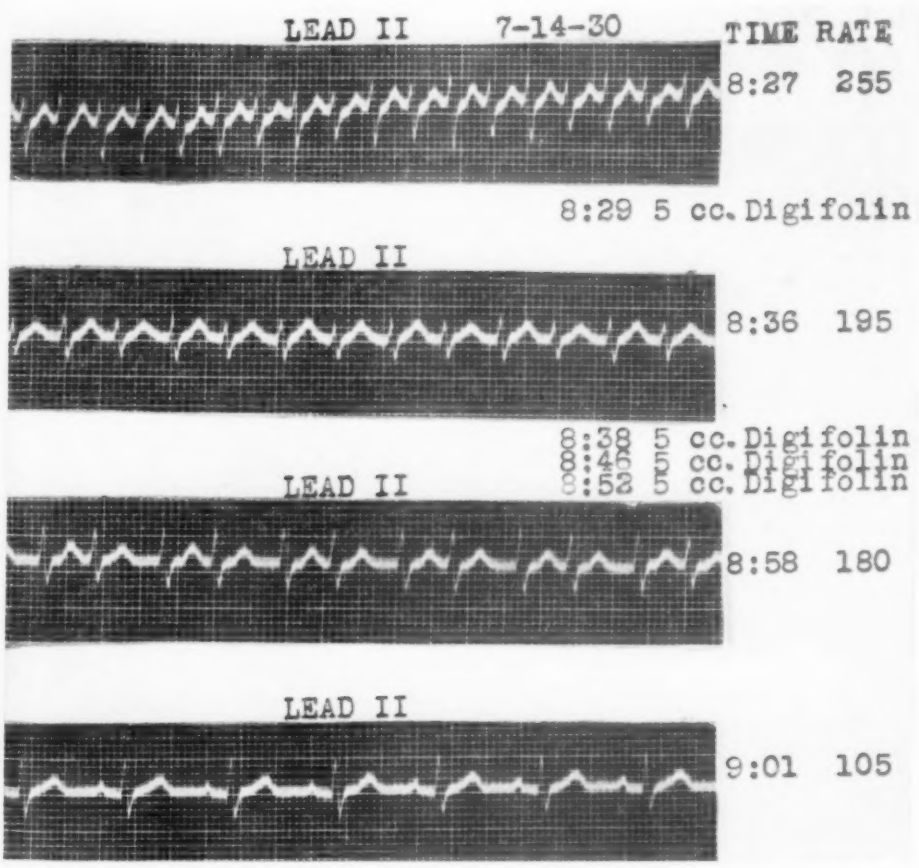

FIG. 3. Nodal tachycardia. Digitalis. Tracings every three minutes; string under constant vision.

tachycardia, the rate being 180. Three doses of 4 cc. each of digalen were given by vein in thirty-three minutes. After the first dose there was a gradual slowing so that after fifty minutes the rate had dropped to 125, but nodal rhythm persisted. Seven minutes later there was a sudden drop of rate to 50, irregular, but of sinus mechanism. (See figure 5.)

to effect an arrest. Since his first admission he has been in tachycardia except when under digitalis. It took about 25 to 30 cc. of the tincture to arrest an attack, but the tachycardia reappeared in about five days if digitalis was left off. Large maintenance doses were required and even then after a few days a fast sinus arrhythmia with relapses to auricular tachycardia occurred.

After leaving off digitalis for three weeks he was admitted in an attack, rate 170, of two weeks duration. Eight-tenths of a gram of quinidine sulphate was given in divided doses by vein over a period of one and one-fourth hours. Though unmistakable evidence of intoxication was present, sinus mechanism was not established; but the rate was slowed. Vagal stimulation then caused

After leaving off digitalis for a month he again entered the hospital in auricular tachycardia, rate 145, duration three weeks. After exercise the rate rose to 160. He was then given atropine, 1/25 grain. During the next thirty-seven minutes he was given 8 cc. of digalen and 4/150 grain of atropine. Ten minutes later he had a sinus mechanism, but in fifteen minutes more this had changed

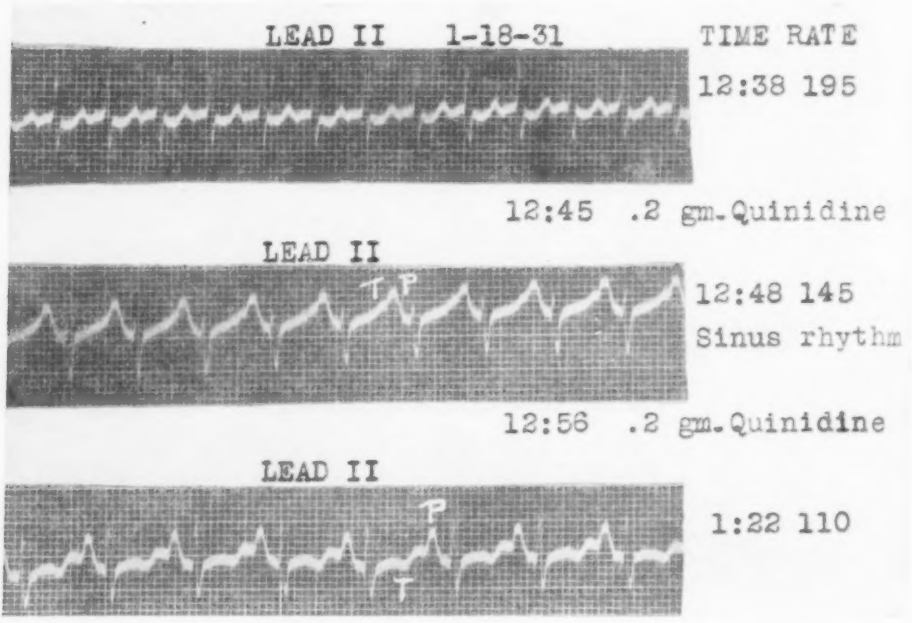

FIG. 4. Nodal tachycardia. Quinidine sulphate. Tracings three to seven minutes; string constantly observed.

no intermission, though previously it had. (See table I.)

The next day with a rate of 170 a total of 16 cc. of digifolin was given by vein in divided doses during a period of thirty-four minutes. Slowing of the rate began early, and in thirty-six minutes sinus mechanism ensued with the fairly slow rate of 95. The change in rate was so gradual that it was impossible to tell from movement of the string when the change of mechanism took place, though the patient knew when it occurred by the relief of palpitation. (See figure 6.)

back to an auricular rhythm which persisted until the next day, when he had a slow sinus rhythm. There can be no doubt but that he had enough atropine to block the vagus and digitalis enough to cause full effect. (See figure 7.)

COMMENT

In two of the above three cases in which quinidine was used there was an arrest of the attack. In both the mechanism was possibly nodal. In all three there was a definite slowing of

TABLE I

The effect of administering quinidine sulphate in Case IV.

E.C.G.	TIME	MEDICATION	HEART RATE	REMARKS
1.	7:48 P.M.		170	
	8:00 P.M.	0.2 grams quinidine intraven.		
2.	8:30 P.M.		160	
	8:35 P.M.	0.2 grams quinidine intraven.		
3.	8:42 P.M.		150	
	8:52 P.M.	0.2 grams quinidine intraven.		
4.	8:57 P.M.		150	
	9:16 P.M.	0.2 grams quinidine intraven.		
5.	9:22 P.M.		150	
6.	9:27 P.M.		150	Auricular tachycardia persists
7.	9:46 P.M.		150	

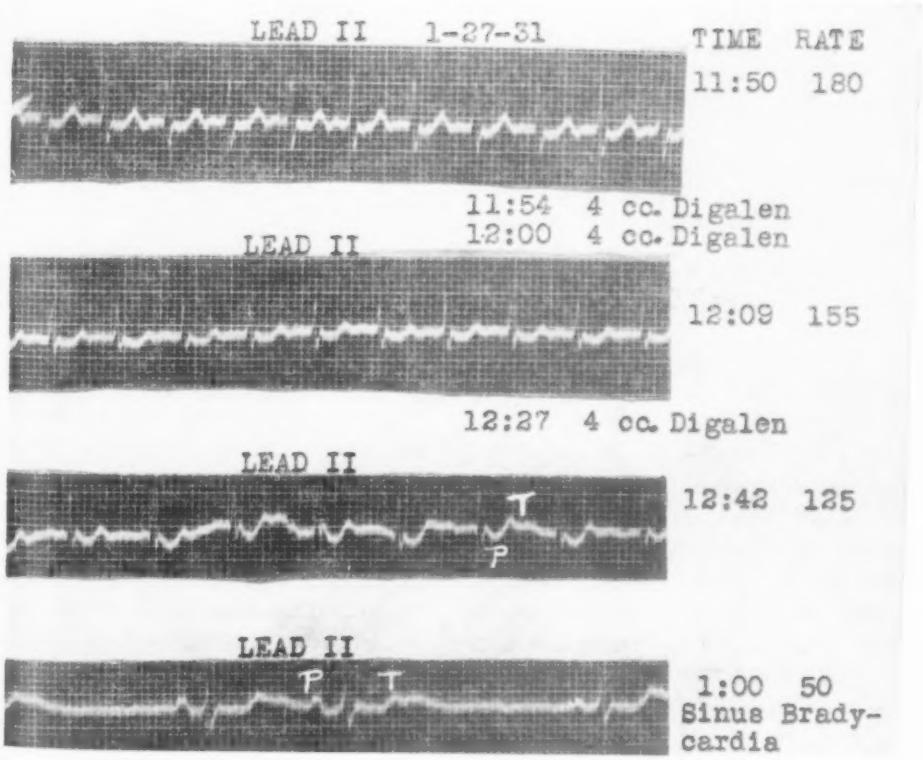

FIG. 5. Nodal tachycardia. Digitalis. Tracings every five minutes; string observed constantly.

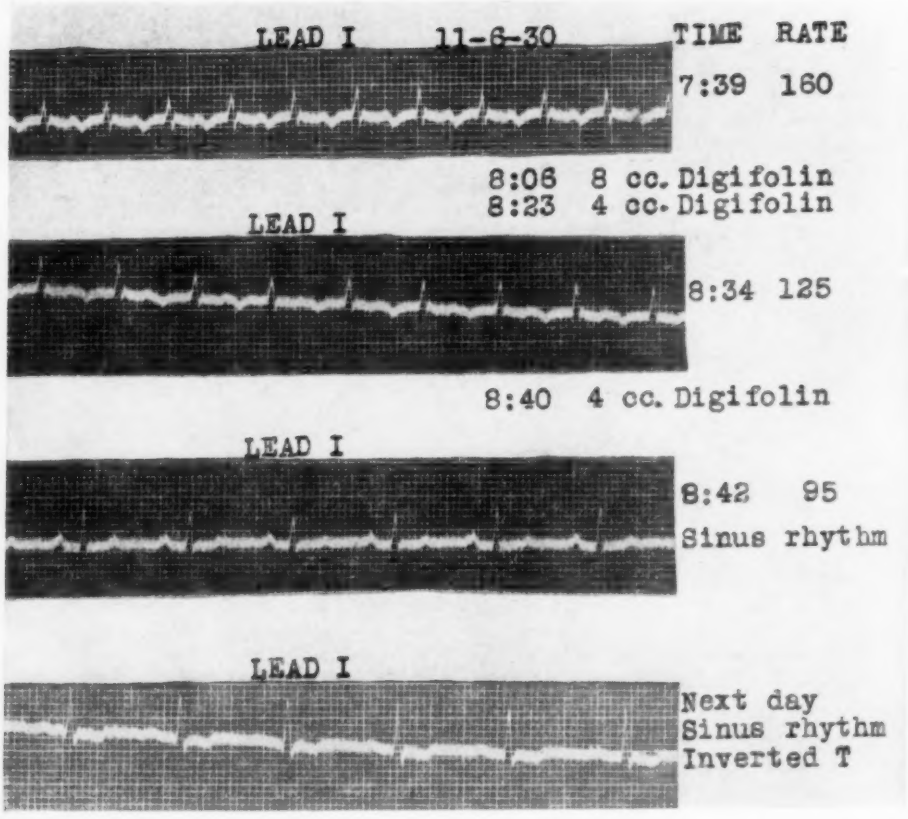

FIG. 6. Auricular tachycardia. Digitalis. Tracings every one to six minutes.

the rate of the attack. In the two in which the attack was arrested, the sinus rate was at first almost as fast as the paroxysmal rate. Assuming that the mechanism of paroxysmal tachycardia is a circus movement, quinidine slows the rate by slowing conduction and arrests the attacks by increasing the refractory period of the auricular muscle. In the two cases reacting to quinidine, in one in which quinidine was inactive, and in one in which quinidine was not used, digitalis slowed the rate and arrested the attack as did quinidine. It is difficult to explain the action of digitalis in slowing the rate for in other examples of circus movement, namely flutter, it increases the rate. By its effect through the vagus, digitalis may decrease the conduction time out of proportion to the decrease in the refractory period and end circus movement, or it may end circus movement by its effect on the muscle in lengthening the refractory period.

In one case digitalis failed to arrest permanently an attack after complete paralysis of the vagus by atropine. It would seem, therefore, that digitalis arrests the attacks by action on the vagus. With atropine, digitalis slowed the auricular rate.

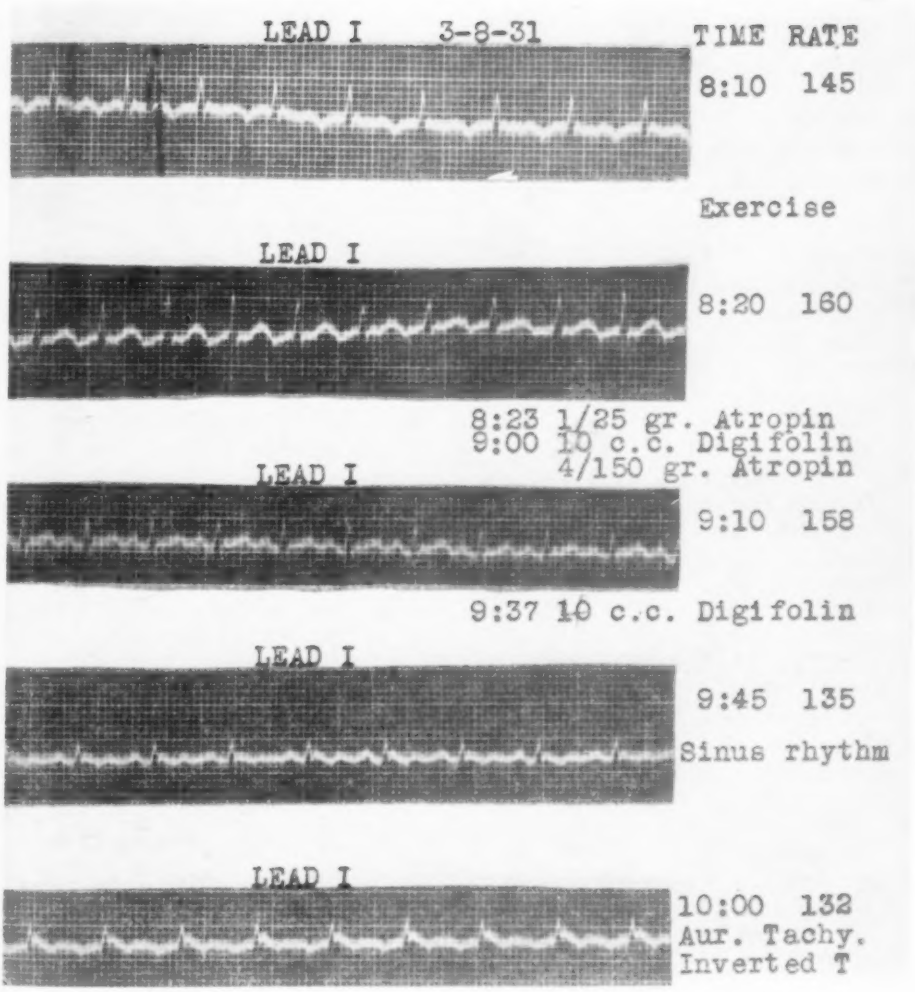

FIG. 7. Auricular tachycardia. Digitalis after atropine.

SUMMARY

The important features in a series of twenty-six cases of supraventricular tachycardia are noted. Cases of unusual duration are reported, one case having an attack lasting three years, another being in a permanent attack for nearly six years unless treated.

Quinidine in most cases of supraventricular tachycardia is the more desirable drug for arresting an attack, though in the presence of heart failure digitalis will act and may be the drug of choice. In two cases where quinidine was continued for a year, it had to be used in increasing doses.

Continuous digitalization has been extended over periods as long as one and a half years. While the continuous use of digitalis is to be preferred over

quinidine, the drug must be used in such large doses that toxic effects appear.

Both digitalis and quinidine will slow the rate and arrest the attacks in the same patient. Until the mechanism of these attacks is better known, the mode of action of drugs cannot be explained.

Digitalis did not arrest an attack in a patient who had received 1/15 grain of atropine.

Both digitalis and quinidine act well by mouth, and few cases need the drugs by vein.

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Answer "Yes" or "No"

"NATURALLY, from the very nature of its objective, the hypothetical question must be a biased and one-sided affair in order to be of value as a piece of evidence carrying weight with those deciding the case. It is just this one-sidedness which frequently taxes the physician-witness's conscience in fairly answering by the generally requested 'yes' or 'no'. . . . . No medical expert can be compelled to answer either positively or negatively a hypothetical question that is based either wholly or in part upon subject matter so arranged that he cannot conscientiously give the reply asked for. Furthermore, a medical expert's answer to the effect that he does not know, is also expressing an opinion, and is considered a reasonable and logical reply thereto. The lawyer, to be sure, usually requests hypothetical questions to be answered in the affirmative or negative by monosyllables; *but it nevertheless remains the inalienable right of the witness to testify according to the dictates of his conscience in harmony with his oath as a witness, and not at all according to the preconceived notions of a lawyer propounding a debatable question.*"

(From *Medical Jurisprudence* by Carl Scheffel, J. Blakiston's Son and Company, Philadelphia, 1931.)

The Response of the Cardiovascular System to Respiratory Strain: A Measure of Myocardial Efficiency*

By Allan Eustis, B.S., M.D., F.A.C.P., *New Orleans, La.*

THE importance of determining the functional capacity of the myocardium in organic heart disease has engaged the attention of cardiologists for many years; but in 1922, Brittingham and White,[1] after a careful study of the tests then in use, concluded that there was no satisfactory test for cardiac function. During this same year Frost[2] arrived at the same conclusion, and at the annual meeting of the Association of Life Insurance Medical Directors of America, he described a new cardiorespiratory test developed in collaboration with Dwight, which was being used by the New England Life Insurance Company in evaluating cardiac cases. A report of this meeting was not published in the current medical journals which probably accounts for its being overlooked by the profession at large, but subsequent publications by Frost[3] should have augmented the scant attention which it has received, especially at the hands of the cardiologists; although Herrmann[4] in 1927 states, "The cardiorespiratory test as described by Frost is certainly a most promising clinical method of estimating the functional state of the circulatory system". Several of my publications[5] bear evidence that its clinical value has been appreciated by me since 1924, and at the Sixth Annual Congress of Anesthetists in Washington in May, 1927, I suggested that a simplification of Frost's original technique, utilizing only steps 6, 7 and 8, would result in a simple test whereby anesthetists could judge the patient's cardiovascular state before an anesthetic was given. Later, I published[6] a description of the modified technic, which I have been using for the past five years.

In following the progress of cases of chronic myocardial insufficiency the cardiorespiratory test has been of inestimable value to me, while the therapeutic indications have often been predicated upon the patients's response to this test, as well as the diagnosis being suggested by same. Common sense should prevail in the interpretation of the response, as Schmitz[7] so aptly suggests in a resumé of the value of various heart function tests, when he calls attention to the fact that most investigators have made the error of employing the various tests as heart tests rather than as tests of the person with

*Presented at the Baltimore meeting of the American College of Physicians, March 24, 1931.

heart disease. The cardiorespiratory test depends upon the physiological fact, that increase in intrathoracic pressure caused by forcible expiration, results in a rise in systolic blood pressure, a subsequent fall, and then a rise above that of the initial systolic blood pressure in an individual with a normal cardiovascular system.

A study of 160 responses on 125 cases taken from my office files, forms the basis of this paper, no attempt being made to review other cardiac functional tests. However, attention should be given to a recent combination test devised by Mackenzie[8] and his coworkers in the Medical Department of the Prudential Life Insurance Company, which is called the flarimeter test, this being an ingenious combination of the step test, cardiorespiratory test, and the ability to hold the breath after exercise. I have had but little personal experience with this test, but it appears too complicated for general clinical use. Yet, the spirometer used in the test for expiration is probably preferable to the simplex spirometer, which as an accurate measure of vital capacity, is admittedly open to criticism.

A letter recently received from Frost, who is Medical Director of the New England Mutual Life Insurance Company, where the cardiorespiratory test has been in use in his department since 1922, will bear quoting in full. He writes:

"We are still using the cardiorespiratory test routinely in our work and think as much of it as ever. Our experience is now based on from 8,000 to 10,000 such tests. We have just finished investigating 5,000 individuals whom we have accepted upon the basis of the test and are satisfied that it provides us with the means of controlling the mortality in the circulatory cases at least for a period of eight years, which is, of course, the period of our present study. We still believe that it gives us an idea of the ability of the circulation at the time of examination which we can obtain in no other way. You may be interested to know that in the 5,000 cases which we have just investigated, the gross mortality of the group was 42 per cent of the American Experience Table. Analyzing this material more carefully, we find that in all the cases in which the reaction of the test has been a straight normal we have a mortality of 30 per cent. On the other hand, in those cases in which the reaction tended to abnormality, in particular, of the hyperactive type, the mortality mounts to 75 per cent. In other words, with an average mortality of 42 per cent, in the presence of a perfectly straightforward, normal cardiorespiratory test, we have a mortality of 30 per cent and in the presence of the tests bordering upon abnormality we have the higher mortality of 75 per cent. From our point of view, therefore, basing our results on mortality figures, our experience has been entirely satisfactory and so far as we know better than any hitherto obtained."

The modified technic of the cardiorespiratory test which I have adopted and used extensively for the past five years is as follows: After physical examination of the patient and recording of results, the systolic and diastolic pressure is taken by the auscultatory method with the patient seated, the pulse rate being counted at the same time by auscultation over the brachial artery. The pressure in the cuff is then released and the patient told to expire through the spirometer after full inspiration, cautioning him to watch the pressure gauge and keep the pressure uniform at 20 mm. of mercury. The systolic pressure is taken before the patient inhales, and after expiration has been completed, the maximum systolic pressure being recorded. With-

PROTOCOL OF CASES

No.	Date	Age	Diagnosis	Pulse		Blood Pressure		Response	Base Line	Cap'ty Blown	Comment
				Before	After	Before	After				
1	5/13/26	54	Chr. myocarditis	68	72	118/60	118/70	10–10–15	Stationary	285-270-265	Poor
2	8/25/29	57	Myocarditis, angina pectoris	80	80	125/80	125/80	10– 5–10	Falling	100- 90-100	Poor
3	10/21/29	40	Myocarditis	80	80	140/85	148/90	10–15–20	Stationary	120-130-120	Fair
4	5/15/30	48	Chr. valvular heart disease	72	76	135/90	140/90	30–45–55	Ascending	280-280-270	Good
5	3/31/30	75	Chr. myocarditis	68	64	160/90	164/90	10–15–10	Stationary	140-160-160	Poor
6	10/ 9/29	52	Myocardial insufficiency	84	84	135/90	135/90	15–20–25	Ascending	200-200-200	Fair
7	10/21/28	43	Diabetes mellitus, myocard. insuff.	80	96	120/80	120/80	10–15–18	Stationary	160-160-170	Poor
8	5/20/30	56	Carcinoma of duodenum	120	136	145/100	160/100	30–40–40	Ascending	200-210-200	Good
9	2/28/31	49	Chronic valvular heart disease	84	90	116/76	120/80	24–36–44	Ascending	180-180-190	Good
10	5/ 2/30	58	Chr. myocarditis	76	84	135/90	135/90	15–25–25	Ascending	180-170-180	Fair
11	4/ 1/30	59	Fibroid tbc. lungs, duodenal ulcer	76	72	105/65	105/70	10–15–15	Stationary	160-160-160	Poor
12	3/26/30	32	Chr. myocarditis, chr. tonsillitis	118	124	144/90	144/90	20–40–50	Ascending	160-160-160	Good
13	3/10/30	30	Myocardial insufficiency	76	80	115/80	112/80	10–15–15	Falling	200-200-200	Poor
13b	4/22/30	30	Myocardial insufficiency	80	80	110/75	112/70	15–20–30	Ascending	220-240-230	Good
14	4/16/30	51	Myocardial insufficiency	60	68	110/70	110/70	0–4—2	Stationary	160-180-160	Poor
15	8/10/29	47	Myocardial insufficiency	80	80	125/80	118/80	15–10–10	Falling	200-200-210	Poor
16	1/27/30	47	Ch. cholecystitis, myocarditis	84	84	120/80	122/80	10–20–30	Ascending	220-240-230	Fair
17	3/24/30	55	Myocardial insufficiency, cholelithiasis	76	76	116/90	120/90	14–20–20	Ascending	170-200-200	Fair
18	8/28/29	18	Chr. valvular heart disease	120	120	120/80	120/80	20–25–30	Ascending	180-180-180	Good
19	10/ 1/29	58	Hypertension	64	64	170/90	160/90	15–25–35	Stationary	180-180-180	Good
20	12/11/28	47	Myocardial insufficiency	96	104	135/90	120/90	5– 5–10	Falling	180-190-200	Poor
21	3/16/28	52	Myocarditis, dilatation	78	84	150/140	140/130	10–15–10	Stationary	60- 70- 65	Poor
21b	4/23/28	52	Myocarditis, dilatation	88	88	124/80	128/80	6– 6– 6	Stationary	80- 80- 80	Poor
21c	6/ 7/28	52	Myocarditis, dilatation	84	84	135/80	135/80	10–15–20	Ascending	60- 60- 65	Fair
22	4/ 2/29	52	Myocardial insufficiency	68	68	120/80	120/80	5–10–15	Stationary	180-160-160	Poor
23	10/ 3/28	52	Cardio-renal disease	84	90	165/90	170/90	15– 5–15	Ascending	100-120-100	Poor
24	4/ 9/29	60	Myocarditis, dilatation	68	68	140/80	140/80	10–15–20	Ascending	150-150-150	Fair
25	8/ 4/28	18	Myocarditis, sinus arrhythmia	76	80	120/80	115/80	25–40–40	Ascending	200-200-200	Good
25b	9/ 4/28	18	Myocarditis, sinus arrhythmia	80	84	110/70	120/80	25–30–30	Ascending	185-215-215	Good
26	9/15/28	48	Infectious arthritis	80	80	108/60	98/60	20–10–20	Stationary	150-160-160	Poor
27	1/ 9/29	21	Neuro-circulatory asthenia	80	80	108/70	110/70	7– 7–10	Stationary	120-110-115	Poor
28	1/28/28	67	Cardio-renal disease, diabetes mellitus	76	80	145/100	145/100	10–15–25	Stationary	120-120-120	Fair
28b	12/24/30	67	Cardio-renal disease, diabetes mellitus	76	76	150/90	155/90	15–20–20	Stationary	130-130-140	Fair
29	6/ 8/28	23	Chronic myocarditis, dilatation	84	68	108/80	100/80	7– 3–10	Falling	30- 30- 35	Poor
30	8/ 3/27	58	Chronic myocarditis	80	84	120/80	120/60	40–30–10	Stationary	310-310-310	Poor
31	12/ 3/26	38	Chr. cholecystitis, chr. arthritis	80	68	112/80	120/90	16– 4– 6	Ascending	140-150-160	Poor
32	4/11/27	58	Obesity, chr. prostatitis	68	68	118/70	172/70	20–30–35	Ascending	225-210-210	Good
32b	8/ 2/28	59	Obesity, chr. prostatitis	80	84	130/80	135/80	15–20–30	Ascending	120-140-120	Good
33	2/18/27	43	Chronic myocarditis	84	84	85/60	85/60	25–25–25	Ascending	240-240-225	Fair
34	6/20/24	55	Chronic cholecystitis	80	80	130/80	130/80	6– 6–20	Stationary	275-270-270	Poor
35	2/ 1/28	9	Chr. valvular heart disease Chr. myocarditis	108	96	95/45	90/40	20–28–28	Stationary	80- 90- 90	Fair
36	7/21/26	20	Post-influenzal myocarditis	80	88	112/70	108/70	22–10– 2	Descending	90-100-100	Poor
37	5/ 4/27	44	Mediastinal adenitis	88	84	110/60	116/70	14–25–30	Ascending	195-190-195	Good
38	5/29/28	60	Neurasthenia	76	76	130/80	138/80	25–30–40	Ascending	160-170-180	Good
39	9/10/27	25	Hypotension, pregnancy	76	76	105/80	105/80	10–15–20	Stationary	225-225-225	Fair
40	11/ 6/29	36	Chronic nicotine poisoning	84	88	135/90	140/90	15–25–35	Ascending	220-230-230	Good
41	3/13/28	56	Chronic myocarditis	72	72	142/100	130/90	2– 2– 4	Falling	160-160-180	Poor
42	3/19/26	42	Chronic cholecystitis	100	84	150/100	170/100	20–30–50	Ascending	180-180-180	Good
42b	2/18/27	43	Chronic cholecystitis	72	80	140/90	150/90	10–20–40	Ascending	130-120-180	Good
43	4/19/26	47	Cardio-renal disease, hypertension	103	120	220/110	220/130	20–30–30	Ascending	170-170-170	Fair
44	11/18/27	53	Chr. interstitial nephritis Hypertension	65	60	185/120	190/130	15–25–55	Ascending	160-140-180	Good
45	5/ 2/28	58	Chronic myocarditis	80	80	100/60	100/60	10–20–30	Ascending	200-220-220	Good
46	3/ 4/27	64	Carcinoma of lungs	72	80	110/100	110/70	18–20–30	Ascending	150-150-135	Fair
47	10/12/26	56	Chronic myocarditis	105	96	118/70	120/110	20–20–20	Ascending	240-240-230	Fair
			Chronic myocarditis	84	84	150/80	150/80	20–25–20	Stationary	210-210-215	Fair

46	3/ 4/27	64	Carcinoma of lungs	72	80	110/100	110/70	18–20–30	Ascending	150-150-135	Fair
47	10/12/26	56	Chronic myocarditis	106	96	118/70	120/110	20–20–20	Ascending	240-240-230	Fair
48	5/12/28	60	Chronic myocarditis	84	84	150/80	150/80	20–25–20	Stationary	210-210-215	Fair
49	5/ 5/27	47	Cirrhosis liver, chr. myocarditis	112	80	88/50	88/60	2– 8– 8	Falling	105-105- 90	Poor
50	6/19/24	50	Fibrosis of lungs	80	80	160/90	160/90	10–20–30	Stationary	260-300-345	Good
51	7/26/27	71	Chronic myocarditis	72	72	110/60	105/60	10– 5– 5	Falling	195-190-180	Poor
52	12/15/25	55	Chronic aortitis	64	72	128/80	140/80	15–28–30	Ascending	315-315-315	Fair
53	12/16/27	62	Chr. myocarditis, hypertension	80	80	160/80	140/80	5– 0– 0	Falling	120-140-140	Poor
54	4/ 1/26	41	Chronic cholecystitis, chr. myocarditis	64	72	128/80	140/80	30–20–30	Ascending	300-300-300	Good
55	11/25/25	52	Hypotension	88	88	92/60	98/70	24–18–20	Stationary	300-315-315	Fair
56	5/ 6/27	58	Hypothyroidism	76	76	98/60	100/60	10–20–35	Ascending	90-105-105	Good
57	12/ 3/27	66	Chronic prostatitis, myocarditis	76	76	210/90	200/90	20–10–10	Stationary	140-130-120	Poor
58	7/11/27	43	Chronic cholecystitis and appendicitis	76	76	130/80	128/80	20–40–60	Ascending	300-300-315	Good
59	4/ 2/27	42	Hyperchloridia	78	78	128/80	128/80	20–20–20	Stationary	168-190-200	Fair
60	8/ 4/26	57	Post-influenzal myocarditis	64	68	138/80	140/80	6– 6–20	Falling	255-255-255	Poor
61	5/29/28	44	Obesity	76	76	135/90	135/90	15–20–35	Ascending	200-200-200	Good
62	2/26/27	47	Routine physical examination	72	72	130/80	135/80	30–40–50	Ascending	285-285-285	Good
63	9/ 5/27	32	Infectious arthritis	80	84	108/70	110/70	10–16–20	Ascending	160-160-160	Fair
64	9/ 6/27	56	Chr. cholecystitis, chr. myocarditis	76	76	106/60	108/60	20–30–35	Ascending	165-165-165	Good
				72	72	110/60	110/80	10–20–30	Ascending	210-230-240	Good
65	2/15/28	30	Paroxysmal tachycardia	78	80	195/110	180/100	10–10–10	Stationary	150-165-165	Poor
66	4/18/27	77	Dilatation of heart, chr. myocarditis					(5″ before appearance)			
67	11/30/27	75	Cardio-renal disease, dilatation of heart	80	80	210/100	160/80	−10;−30;−70*	Falling	120-100-100	Poor
68	2/25/27	58	Partial heart-block	76	80	132/80	135/80	18– 8–28	Ascending	180-190-190	Fair
69	11/14/27	49	Hypertension, chronic myocarditis	76	84	180/150	180/140	20–30–20	Stationary	180-180-160	Fair
70	1/15/29	49	Chronic myocarditis	84	84	135/90	135/90	15–25–35	Stationary	180-180-180	Good
71	2/ 4/29	39	Chr. valv. heart disease, card dilat.	76	76	130/75	130/80	5– 0– 0	Falling	95-100-100	Poor
72	9/ 5/28	58	Chr. cholecystitis, chronic myocarditis	84	84	150/80	135/80	0–10–20	F'l'g(20m.)	120-110-100	Poor
73	7/19/27	53	Chronic valvular heart disease	76	76	145/90	148/100	15–25–45	Ascending	210-250-250	Good
73b	3/18/29	55	Chronic valvular heart disease	88	84	170/100	170/100	25–30–40	Ascending	180-180-175	Good
74	8/17/28	46	Chronic myocarditis, dilatation of heart	108	104	190/140	185/170	20–30– 0	Falling	140-140-140	Poor
75	9/ 5/28	42	Abscess of lungs, dilatation of heart	76	76	85/40	90/40	15–25–15	Stationary	180-180-195	Poor
76	12/30/25	34	Acute glomerulo-nephritis, hypertension	80	80	154/100	160/100	10–20–30	Ascending	170-180-190	Good
77	5/28/28	50	Chronic myocarditis	68	76	150/100	150/100	20–25–30	Ascending	120-120-120	Fair
77b	8/ 1/28	50	Dilatation of heart	76	80	170/90	170/90	10–20–25	Stationary	120-130-120	Poor
78	4/ 2/29	17	Neuro-circulatory asthenia	112	124	108/80	118/80	20–30–40	Ascending	80- 80- 80	Good
79	2/ 8/29	29	Neuro-circulatory asthenia	84	80	108/80	108/70	2– 2– 2	Stationary	190-190-190	Poor
80	4/17/28	43	Chr. myocarditis, mediastinal adenitis	92	92	145/95	145/90	25–30–35	Ascending	180-180-160	Good
80b	11/ 2/28	43	Chr. myocarditis, mediastinal adenitis	100	96	135/80	130/80	20–20–25	Stationary	140-150-150	Fair
81	3/18/29	60	Hypertension	88	80	160/120	170/120	20–30–40	Ascending	180-180-180	Good
82	8/22/28	40	Dilatation of heart	64	84	118/110	108/80	8– 8–10	Falling	150-160-150	Poor
83	2/ 4/29	39	Paroxysmal tachycardia	94	100	134/80	140/80	20–30–45	Ascending	110-100-120	Good
84	4/ 6/29	51	Hypertension	76	72	176/120	180/120	20–20–30	Ascending	160-160-180	Good
85	3/ 5/29	61	Chronic cholecystitis	80	84	170/120	170/110	20–30–40	Stationary	130-140-130	Good
86	11/27/28	48	Chr. myocarditis, chr. cholecystitis	84	84	120/80	120/80	20–40–48	Ascending	160-160-160	Good
86b	8/27/29	49	One year after cholecystectomy	84	76	100/60	110/70	22–35–38	Ascending	190-220-210	Good
87	11/14/28	64	Myocardial insufficiency	48	52	130/80	120/80	10– 5– 5	Falling	170-175-180	Poor
88	11/ 7/27	51	Myocarditis, hypertension	80	76	160/100	150/100	10– 0– 0	Falling	80- 90- 75	Poor
89	2/ 2/26	50	Neuro-circulatory asthenia	68	76	118/70	124/80	10–15–20	Ascending	285-285-270	Fair
90	5/10/27	38	Myocardial insufficiency	96	96	130/80	130/80	10–30–30	Stationary	225-240-195	Fair
90b	6/ 2/27	38	Myocarditis, compensation	80	80	120/80	128/80	30–40–45	Ascending	270-285-285	Good
90c	3/ 4/31	42	Physical examination	88	76	125/80	128/80	15–25–35	Ascending	180-180-180	Good
91	1/11/27	39	Myocardial insufficiency	84	88	126/80	124/80	30–30–20	Stationary	255-255-255	Fair
91b	3/ 2/27	39	Myocardial insufficiency	76	76	118/70	104/70	12–22– 2	Falling	285-295-285	Poor
91c	4/21/27	39	Myocardial insufficiency	76	76	128/80	118/70	20–18–10	Stationary	240-270-260	Poor
91d	6/ 3/27	39	Myocardial insufficiency	72	72	115/70	110/70	5–10– 0	Falling	300-210-210	Poor
91e	10/ 4/27	39	Myocardial insufficiency	76	76	130/80	128/80	10–20–25	Ascending	180-180-200	Fair
91f	12/ 5/27	39	Myocardial insufficiency	68	76	118/70	118/70	10–20–30	Ascending	180-240-235	Good
91g	2/19/31	43	Myocardial insufficiency	76	76	138/90	135/90	10–15–20	Ascending	220-240-245	Fair

PROTOCOL OF CASES—Continued

No.	Date	Age	Diagnosis	Pulse		Blood Pressure		Response	Base Line	Cap'ty Blown	Comment
				Before	After	Before	After				
92	11/26/28	61	Myocarditis, hypertension	68	76	170/110	150/90	10–10–10	Stationary	140-140-140	Poor
92b	5/19/30	62	Myocarditis, hypertension	70	76	170/100	170/100	30–40–45	Ascending	160-160-160	Good
92c	2/26/31	63	Myocarditis, hypertension	76	76	165/100	175/100	20–35–45	Ascending	140-140-140	Good
93	11/27/25	55	Adhesive pericarditis, myocarditis	120	112	118/80	110/80	12–12– 0	Stationary	180-180-180	Poor
93b	2/ 4/26	56	Adhesive pericarditis, myocarditis	112	92	118/80	124/80	10–20–25	Ascending	165-165-165	Fair
93c	2/28/31	61	Adhesive pericarditis, myocarditis	104	112	120/90	120/90	18–15–18	Stationary	80- 90-100	Poor
94	1/20/28	58	Myocarditis, angina pectoris	80	120	140/90	140/90	20–20–20	Ascending	160-180-180	Fair
95	11/30/27	48	Myocarditis, angina pectoris	72	76	108/60	104/60	40–30–20	Stationary	220-220-220	Fair
96	11/ 3/26	57	Hypertension	84	90	225/125	215/125	10–20–40	Stationary	240-240-240	Good
97	9/28/28	48	Hypertension	88	88	210/150	215/160	30–35–40	Ascending	230-240-250	Good
98	2/ 6/26	53	Hypertension, hypertrophy of heart	84	96	190/140	215/160	5– 5–10	Stationary	215-225-230	Poor
99	3/17/26	46	Hypertension, pyonephritis	100	100	170/120	170/120	20–20–40	Stationary	270-275-285	Good
99b	9/26/28	48	Hypertension, decompensation	80	84	170/110	165/110	10–15–20	Stationary	200-190-190	Fair
99c	3/ 6/31	51	Hypertension, compensation	64	68	190/120	220/130	30–50–	Ascending	160-160-	Good
100	4/ 5/29	51	Hypertension	88	84	174/110	194/110	12–26–46	Ascending	140-160-160	Good
101	5/ 3/27	48	Hysteria	76	76	110/60	120/60	20–30–40	Ascending	225-240-240	Good
102	12/15/24	38	Asthma	76	92	130/90	136/90	40–40–60	Ascending	155-165-180	Good
103	5/ 6/25	34	Neurasthenia	90	100	118/70	120/70	10–14–20	Ascending	105-165-180	Fair
104	4/22/29	50	Chronic cholecystitis	80	88	140/90	140/90	10–20–30	Stationary	200-200-200	Good
105	1/23/28	74	Myocarditis, dilatation of heart	64	84	120/100	120/100	10–10–10	Stationary	100- 80- 80	Poor
105b	2/25/28	74	Myocarditis, dilatation of heart	76	80	130/80	135/80	10–15–20	Stationary	120-135-120	Fair
105c	5/ 3/28	74	Myocarditis, dilatation of heart	74	80	120/80	125/80	10–15–20	Ascending	140-150-150	Fair
106	3/ 1/29	27	Secondary anemia	108	104	118/70	118/70	14–22–30	Stationary	160-160-160	Good
107	10/20/25	50	Myocarditis, hypertension	92	112	160/80	165/90	10–15–35	Ascending	345-345-345	Good
107b	5/26/27	52	Myocarditis, hypertension	92	96	145/90	142/90	40–50–60	Ascending	315-315-315	Good
108	5/30/27	53	Chronic valvular heart disease	76	76	150/90	145/80	5–10– 0	Falling	225-225-225	Poor
109	1/10/30	41	Myocarditis, dilatation of heart	84	88	145/90	145/90	20–20–30	Stationary	160-180-190	Fair
110	5/ 2/30	56	Myocarditis, mediastinal adenitis	96	96	118/70	115/70	12– 7–10	Falling	60- 60- 60	Poor
111	7/ 3/24	43	Chronic valv. heart disease, myocard.	76	74	100/80	100/80	10– 0– 0	Falling	225-255-255	Poor
112	8/29/25	38	Chronic cholecystitis	72	72	120/80	120/80	10–20–30	Ascending	270-270-255	Good
113	1/ 7/40	58	Myocardial disease	68	68	165/90	165/90	5– 5–10	Stationary	220-190-210	Poor
114	10/ 9/28	44	Myocardial disease	76	64	118/80	130/80	15–20–25	Ascending	180-180-200	Fair
115	1/19/31	40	Adherent pericarditis	80	64	115/110	115/110	15–15–15	Stationary	180-180-180	Poor
116	12/12/30	57	Angina pectoris, chronic appendicitis	84	92	145/70	150/70	20–35–45	Stationary	140-160-155	Good
117	7/28/25	55	Chronic myocarditis	80	100	110/70	110/70	10–20– 5	Stationary	90-100-120	Poor
117b	12/ 4/28	58	Chronic myocarditis	72	72	130/80	125/80	10–15–20	Ascending	215-215-215	Fair
117c	2/27/31	61	Chronic myocarditis	76	76	128/80	135/80	25–35–40	Ascending	200-200-200	Good
118	3/ 2/24	54	Myocarditis, dilatation of heart	76	100	150/90	155/90	5–10–10	Stationary	160-160-155	Poor
118b	5/30/27	57	Myocarditis compensation	56	60	165/120	140/80	25–50–40	Ascending	195-210-225	Good
118c	2/20/31	61	Myocarditis compensation	56	64	148/100	165/100	20–30–35	Ascending	180-180-195	Good
119	2/21/29	55	Myocarditis, dilatation of heart	84	84	120/70	115/70	5– 0– 0	Falling	180-180-180	Poor
119b	2/25/31	57	Myocarditis compensation	80	80	130/80	135/80	15–20–35	Ascending	180-200-200	Good
120	2/24/26	62	Myocarditis, dilatation of heart	88	88	110/70	106/70	10–20–20	Ascending	195-195-195	Fair
120b	3/21/27	63	Myocarditis compensation	80	80	130/80	135/80	15–30–40	Ascending	285-285-285	Good
121	4/18/25	48	Paresis of left diaphragm	84	84	130/80	134/80	10–20–30	Ascending	175-175-175	Good
122	7/ 6/27	62	Myocarditis, compensated	76	76	145/90	145/90	15–25–35	Ascending	180-180-195	Good
122b	4/ 3/28	63	Myocarditis, dilatation	120	130	140/90	140/90	10–10– 5	Stationary	120-120-118	Poor
123	3/ 9/25	60	Routine physical examination	64	64	120/75	130/80	10–20–30	Ascending	240-210-225	Good
123b	5/ 5/27	62	Myocarditis, dilatation of heart	76	72	110/80	116/80	10–10– 8	Falling	160-140-150	Poor
124	5/13/29	54	Neuro-circulatory asthenia	72	64	100/65	90/60	10–10– 5	Stationary	180-160-150	Poor
125	12/21/25	31	Chronic nicotine poisoning	80	84	135/90	132/90	14–24–30	Ascending	225-220-240	Good

*Negative response.

out releasing pressure in the cuff, the needle of the spirometer is turned to zero, the systolic pressure is again taken and the patient again instructed to inhale fully and expire through the spirometer as before. Three successive readings are then made, corresponding to steps 6, 7 and 8 in Frost's technic. If it is evident that the patient has not expired his full vital capacity, a second test should be made after cautioning him to inhale and expire fully; or, perhaps only a fourth expiration may be necessary.

The results may be plotted as a curve, the change in millimeters of mercury in blood pressure obtained after each expiration representing the response, and the change in millimeters in the blood pressure obtained just prior to each inspiration representing the base line. A normal response should result in an increase of systolic blood pressure after the third expiration of from 30 to 40 mm. A failure to respond, in my experience, denotes a weakened heart muscle. A falling base line is invariably associated with great dilatation of the heart. The test should not be tried on any individual with marked dilatation of the heart, as I have seen in my early experience a near approach to syncope result in such instances, while in others marked cardiac distress has been observed. The test should be used with caution, also, on patients with a history of anginal attacks, as well as on cases of arterial hypertension. These precautions are needful with any other cardiac function test, but, I believe the danger from the cardiorespiratory test is less than with most cardiac tests involving a strain on the heart muscle.

The test is especially valuable in the study of chronic myocardial insufficiency, and in the protocol, where the diagnosis of myocarditis is recorded, it must be interpreted as the commonly used term to express myocardial degeneration, in which there is usually evidence of myocardial insufficiency. In numerous instances, however, the electrocardiographic tracings denoted myocardial disease, while both clinically and by the cardiorespiratory test, the heart muscle was able to function normally. An examination of the protocol will at once be convincing that the test has been of considerable value to me in clinical evaluation of cases, and a discussion of interesting phases of some cases is in order.

Discussion of Protocol

A study of the protocol shows that of the 160 responses, 56, or 35 per cent, have been listed as poor, i.e., responses below 20 mm.; 40, or 25 per cent, as fair, i.e., 20 to 30 mm., and 64, or 40 per cent, as normal, i.e., above 30 mm. These correspond to the interpretations of Frost as, below normal, low normal, and normal respectively. Further examination will also reveal the fact that some responses were recorded as normal, which on strict interpretation should have been classified as hyperactive, the reason for this being, that I have been particularly interested in detecting weakened heart muscles, rather than evaluating the cases from an insurance standpoint. Of the 56 poor responses, only five, or 2.1 per cent of the total (numbers 22, 26, 34, 87 and 124) failed to exhibit other evidences of a weakened heart muscle after the test. The condi-

* Negative response.

tions present in cases numbers 22, 26 and 34 are those often associated with myocardial changes; while no. 87 was the response of a man 64 years of age, at which time of life a weakened heart muscle is by no means rare. So it is not illogical to suppose that in these cases the poor response was due to weakness of the heart muscle rather than to vasomotor phenomena. No. 124 was obtained on an asthenic individual, who is easily fatigued, with no demonstrable evidence of any focal infection, and with no manifestation of myocardial lesion. His skeletal muscles are flabby, and accepting Starling's idea, that the physiology of the heart muscle as regards its ability to contract and perform work is identical with that of the skeletal muscles, it may be assumed that in this instance, also, the myocardium was weak, though no other symptoms of myocardial insufficiency could be elicited.

In all of the other 51 poor responses, the appearance of a murmur at the apex, or the accentuation of a pre-existing murmur, or an increase in pulse rate, cough or dyspnea were detected after strain was put upon the heart by the test. Some cases (numbers 13, 21, 90, 91 and 92) responding poorly at first examination, responded to the test normally after the heart muscles had been toned up by graduated exercise, and other therapeutic measures. This is especially well illustrated by the responses obtained on case 91 (chart II, D, E and F). Fourteen responses only, on cases of hypertension are recorded, the small number being due to the fact that I rarely run a test on an individual with an initial systolic blood pressure of 180 mm. or over. Such cases tend to respond excessively to the test and the danger of cerebral hemorrhage from hypertension during the test must be constantly borne in mind. Details of a few of these cases will be found in a discussion of chart IV.

Discussion of Charts

Chart I. Responses A, B and C (Case 117). A lawyer, 55 years old, first seen in 1925 (A), weight 202½ lbs.; height 5 ft. 8¼ in. at that time; muscles flabby and pendulous abdomen. There was definite clinical evidence of myocardial insufficiency, but with no organic valvular lesion in the heart. The response to the cardiorespiratory test (A) is characteristic of a flabby heart muscle; a long delay occuring before return of the sounds to the ear after each expiration and a drop in the third step as well as in the base line, with an increase in pulse rate. After removal of diseased tonsils by Dr. Lynch, and gradually increasing exercise, with a loss of 10 lbs. in weight, he had improved symptomatically by 1928, when test (B) was made. While this shows an improvement in the response to the cardiorespiratory test, it is still below normal for a man of his physique. For the past three years he has engaged regularly in golf with progressive improvement physically, his muscles becoming firm with a loss in weight of 14 lbs., while his response (C) to the cardiorespiratory test is normal.

Responses D, E and F (Case 118). A business executive 61 years of age, weight 164½ lbs.; height 5 ft. 7 in., has been under my constant observation since 1912. He had acute dilata-

tion in 1916 which was relieved by one month's rest in bed. Efforts since that time have been devoted to toning up the heart muscle, with the result of gradual improvement in subjective symptoms. In 1924 he still had definite signs of cardiac weakness and his response (D) to the cardiorespiratory test was poor with increase of pulse rate after test. In 1927 there was considerable improvement in clinical symptoms, while the response (E) was better. His vital capacity had increased but weakness of the heart muscle is shown by the drop in systolic blood pressure after the third expiration. At present, there is no clinical evidence of lack of cardiac compensation; he is actively engaged in business and his response (F) to the cardiorespiratory test is normal.

Responses G and H (Case 119). Manager of numerous outdoor amusement concessions, 55 years old; weight 194 lbs.; height 5 ft. 9¾ in., first examined in Feb., 1929. At this time on account of dyspnea and cough and other evidence of dilatation, he was kept in bed for one month. Response (G) to cardiorespiratory test made at initial examination, is very poor with a fall in step 3 and a falling base line. At present there is no clinical evidence of cardiac decompensation, while re-

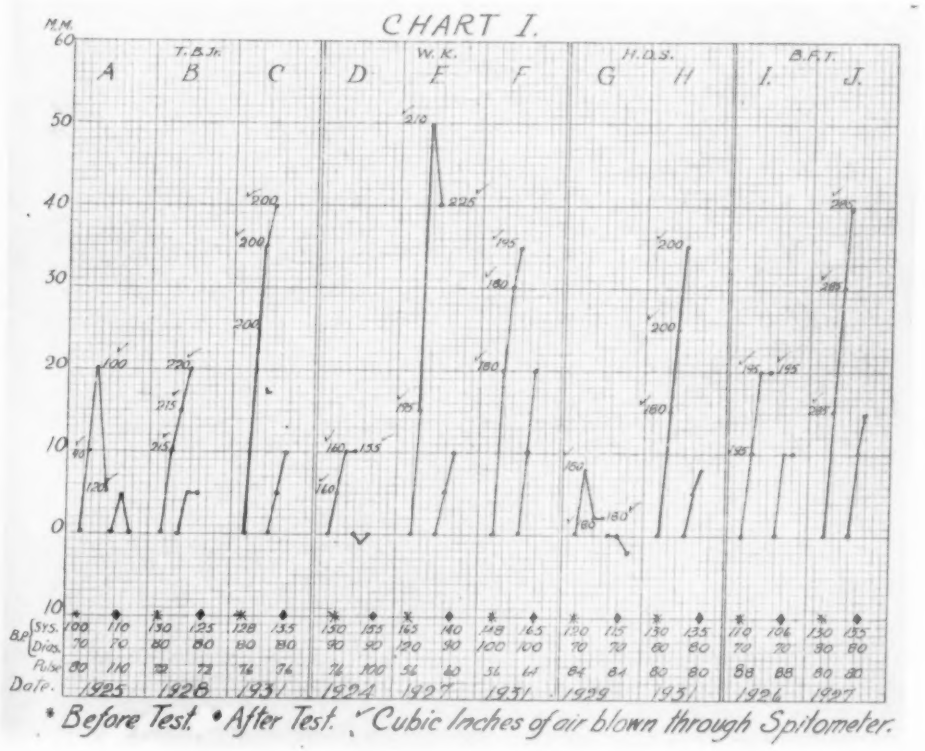

CHART I. Showing improvement in responses to the cardiorespiratory test after compensation has taken place. C, F, H, and I are normal responses (see text).

sponse (H) to the cardiorespiratory test is normal.

Responses I and J (Case 120). An executive 62 years old; weight 185 lbs.; height 5 ft. 8½ in., with clinical evidence of cardiac decompensation following an attack of influenza, gave only a moderate response (I) to the cardiorespiratory test in 1926. After toning up the heart muscle with regular exercise in the form of golf three times a week there was no clinical evidence of cardiac weakness, and his response (J) to the cardiorespiratory test was normal in 1927. He has continued in good health up to the present time.

Chart II. Responses A, B, and C (Case 92). An executive 61 years old; weight 160¾ lbs.; height 5 ft. 8½ in., seen first by me, Nov. 26, 1928, with an enormously enlarged heart, transverse cardiac dullness measuring 20 cm.; extra systoles every third or fourth beat, systolic murmur at apex not transmitted. Enlargement of the liver and slight dyspnea on mounting stairs were the only evidences of an impaired heart muscle, but the response (A) to the cardiorespiratory test with a falling base line gave proof of considerable impairment of the heart muscle. Clinical improvement was prompt and in 1930 the systolic mur-

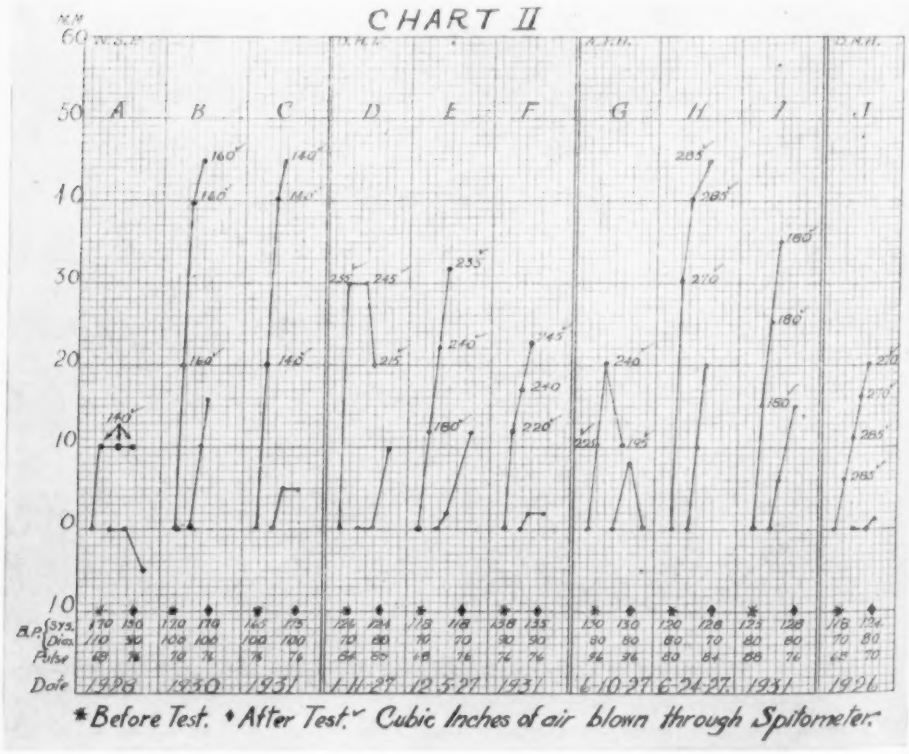

CHART II. Showing varying responses to the cardiorespiratory test. Responses B, C, E, H, and I are normal. F and J are only fair responses (see text).

mur present at the apex in 1928, had disappeared; the transverse cardiac dullness was 16 cm. instead of 20 cm., and the response (B) to the cardiorespiratory test was a high normal one. An examination made in Feb., 1931, showed the heart fully compensated and the response (C) to the cardiorespiratory test was still hyperactive on account of a tendency to hypertension.

Responses D, E, F, and G, H, I represent the type which is often obtained in normal individuals of the obese class, leading sedentary lives, and often showing no symptoms of cardiac decompensation.

Responses D, E, and F (Case 91). An attorney 42 years of age, has been under my observation since 1918, but had never shown any evidence of cardiac weakness until Jan., 1927. He was obese, weighing 262 lbs.; height 5 ft. 10½ in. At this time he began to have extra systoles on slight exertion, which were present after test (D). This response is normal in the first two steps but the drop after third step suggested some myocardial weakness. Gradually increasing exercises under a competent physical instructor, with regulation of his diet resulted in a loss of 30 lbs. by Dec. 5, 1927. At this time there were no extra systoles; physically he was much improved and the response (E) to the cardiorespiratory test was normal. In 1928 he continued his exercise and his weight varied from 232 to 241 lbs. For the past two years he has given up exercise and his weight has gradually increased to 270 lbs. His response (F) to the cardiorespiratory test is low normal.

Responses G, H, and I (Case 90). A capitalist 42 years old; weight 167 lbs.; height 5 ft. 10¾ in., has been under irregular observation since 1915. At college he rowed on the crew but he had taken no regular exercise in recent years. He had "flu" in Dec., 1926. On June 10, 1927 he consulted me for attacks of vertigo, intermittent pulse, and occasional dyspnea. Examination of the heart showed the apex beat 2 cm. outside the nipple line; the transverse cardiac dullness was 17 cm., with an occasional, soft systolic blow audible at the apex but not transmitted. The response (G) to the cardiorespiratory test showed the heart muscle responding normally in steps 1 and 2, but falling in step 3. No medication was given, but he was instructed to play golf daily, increasing gradually the number of holes played. On June 24, 1927, two weeks later, he reported free from symptoms. No murmur was audible, heart hypertrophied and response (H) to cardiorespiratory test was normal. One hesitates to report such improvement in cardiac function in so short a space of time as two weeks, but the frequency with which I have seen this unexpected occurrence in patients taking regular exercise, who formerly, were accustomed to sedentary habits, justifies me. If one accepts Starling's teaching, as aforementioned, that the cardiac muscle does not differ functionally from skeletal muscle, this statement is less incredible. The patient reported for examination on March 4, 1931, stating that he had suffered no further recurrence of cardiac symptoms. His response (I) to the cardiorespiratory test was normal, and physi-

cal examination denoted a normal heart.

Response J (Case 89). A manager of a retail furniture store, 50 years of age; weight 151¾ lbs.; height 5 ft. 6 in., leading a very sedentary life, consulted me Feb. 2, 1926, complaining of forcible cardiac pulsations at frequent intervals. No dyspnea or other symptoms of cardiac decompensation. The transverse cardiac dullness was 14 cm. There was slight roughening of first sound, but no murmurs. No evidence of cardiac pathology could be detected. Response (J) to cardiorespiratory test is feeble and below normal, but is presented as an example of the type of response often obtained in an individual leading a sedentary life. Such a patient continues to present a like response until exercise is enforced. I classify this type as a "lazy heart", and the usual response is probably lacking in clinical significance other than from a prophylactic, or life insurance standpoint. This patient is now in excellent health, still works hard and takes no exercise.

Chart III. These curves represent three cases which have progressed downward clinically, and two fatal cases.

Responses A and B (Case 93). A physician 61 years old; weight 147 lbs.;

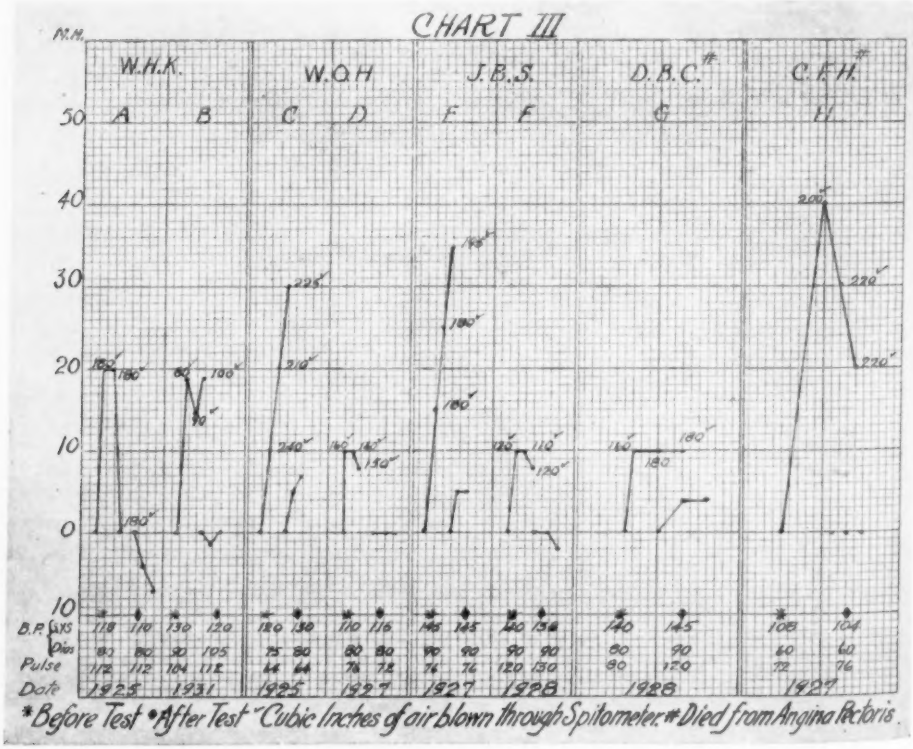

CHART III. Showing responses to the cardiorespiratory test on patients who have retrograded clinically. G and H died from coronary thrombosis (see text).

height 5 ft. 9 in., has been under my observation since 1919, when signs of a failing heart muscle were detected and he was advised to curtail his activities. In 1920 he developed pulmonary tuberculosis and moved to Texas for two years, returning cured of tuberculosis and with an increase in weight of 25 lbs., but with clinical evidence of a failing heart muscle, which ultimately incapacitated him so that he was compelled to retire from the practice of medicine. Electrocardiographic tracings made by Dr. Herrmann in various positions established a diagnosis of an adherent pericardium and chronic myocardial insufficiency. Response (A) in Nov., 1925, was taken while he was still attempting to practice medicine, showing a complete failure after the third step, with the appearance of a systolic murmur at the apex and a severe cough after the cardiorespiratory test. In a few months, decompensation was marked and he consented to remain in bed for two months, which was followed by gradual compensation. A cardiorespiratory test made Feb. 4, 1926, when his heart had clinically compensated gave a response of only 15 mm. (no. 93b in protocol) and the appearance of a systolic blow at the apex which had not been present before the test and which was followed by severe coughing and some dsypnea, demonstrated that compensation was not complete. His heart has compensated fairly well at present and he is able to get out, but slight exertion causes dyspnea and his response (B) to the cardiorespiratory test is still far below normal, while there has been a progressive diminution in his vital capacity.

Responses C and D (Case 123). An executive 66 years old; weight 150 lbs.; height 5 ft. 6¼ in. responded (C) normally to the cardiorespiratory test in 1925 during a routine physical examination, at which time no evidence of any abnormality of the cardiovascular system could be detected. In Sept., 1926, he had acute dilatation of the heart with auricular fibrillation, following influenza, which compensated sufficiently for him to resume his business activities. Cardiorespiratory test (D) made May 5, 1927, showed that the heart had not fully compensated and would not maintain its efficiency under strain. He was advised to curtail his activities, which he refused to do, and since then he has had three attacks of acute dilatation.

Responses E and F (Case 122). An executive 66 years old; weight 232 lbs.; height 5 ft. 11 in., has been under observation since 1915, when he showed signs of beginning failure of the myocardium from obesity. Nine holes of golf daily, on a level course, so toned up his heart muscle that he was free from any signs or symptoms of myocardial insufficiency, and a cardiorespiratory test (E) in July, 1927, gave proof that his heart could withstand strain satisfactorily. During the following winter he took a trip to Nassau, where he ate and drank excessively, taking no exercise and gaining 12 lbs. in five weeks. On his way home, in New York, he contracted a mild afebrile respiratory infection, confining him to bed for only two days, but followed by a cough. Upon reaching New Orleans he noticed that he lacked energy and was slightly dyspneic on mounting stairs. Examination April 2, 1928, revealed an irregular, rapid heart, no

murmurs, but extra systoles and pulsus alternans, while an electrocardiographic tracing made by Dr. Rosen disclosed auricular fibrillation, ventricular extra systoles and evidence of myocardial disease. Cardiorespiratory test (F) at this time showed a poor response with a great reduction in vital capacity since the former test.

Response G (Case 94). A realtor 58 years old; weight 155 lbs.; height 5 ft. 7½ in., consulted me Jan. 20, 1928, for an oppressed feeling in the chest while walking fast, associated with a peculiar tingling sensation in left arm and left hand. There was no actual pain. He had noticed the symptoms for the past two months whenever he walked fast, and he stated that when he stopped the sensation would pass off promptly. There was no enlargement of the heart and no murmurs were audible until he walked briskly up and down the examination room 20 times, when a soft systolic murmur became audible at the apex. The response (G) to the cardiorespiratory test suggested myocardial weakness and he was advised to go to Clifton Springs for treatment of his heart. Unknown to me he consulted a confrere of mine, not telling him of my advice, and the latter, not hearing a murmur, told him that his heart was normal. Three months later he had an attack of coronary thrombosis while walking on the street, followed by dilatation and hypostatic pneumonia, from which he died April 27, 1928. The response to the cardiorespiratory test and the increase in pulse rate, as well as the appearance of a systolic murmur at the apex after the test gave definite evidence of myocardial weakness.

Response H (Case 95). An executive 49 years old; weight 203 lbs.; height 5 ft. 11½ in., a mild diabetic, had been under observation since 1915 and repeated examinations of his cardiovascular system had never shown any abnormality until Nov. 30, 1927, when he complained of a dull, aching pain in the cardiac region; not constant, but coming on usually after walking. The heart was not enlarged, there were no murmurs audible before the cardiorespiratory test (H) was run, which gave a poor response; with appearance, after the test, of extra systoles and a soft systolic murmur at the apex. He was advised to go to bed and rest his heart, but instead, he went to Atlanta to attend a football game, where he had a severe attack resembling angina pectoris. In spite of rest and withdrawal from business activities, he continued to have anginal attacks, and he died from coronary thrombosis Jan. 27, 1930.

Chart IV. This chart is presented to show a tendency to overaction in blood pressure responses in cases of hypertension when the cardiac musculature is compensated.

Responses A, B, and C (Cases 96, 97, and 98). These all died from cerebral hemorrhage in two and one-half years, one and three-quarter years, and five months, respectively, after the tests were made. The poor response (C) of a man 53 years of age, with arteriosclerosis and a history of previous hypertension, denoted a failing heart muscle which coincided with his clinical picture. After compensation of his heart there was a return of hypertension; death following from cerebral hemorrhage.

Responses D and E (Case 99). A practicing physician 51 years of age, a case of hypertension secondary to chronic pyelonephritis, had a compensated heart in 1926 (D); symptoms of beginning loss of compensation in 1928 (E), which disappeared on curtailment of activities. At present he is free from any symptoms of lack of compensation, while response (no. 99c in protocol) denotes that he has full compensation. F, G, H, I, and J are well except for hypertension.

When one realizes that a rise of 70 mm. in blood pressure over an initial blood pressure of 180 mm. results in a systolic blood pressure of 250 mm. the danger of carrying out the test on cases of hypertension with initial blood pressure of 180 mm. or over, is at once manifest and is to be discouraged.

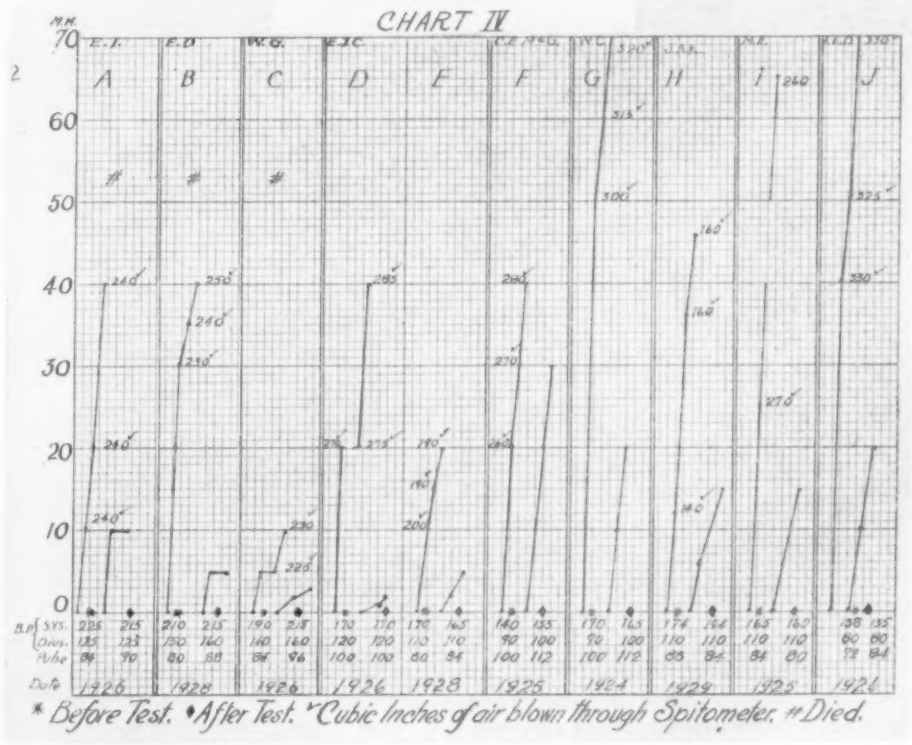

CHART IV. Showing responses to the cardiorespiratory test on cases with hypertension with tendency to hyperactivity. A and B are normal responses. Both died from cerebral hemorrhage. C is a poor response showing weakened heart muscle. After heart had compensated patient died from cerebral hemorrhage. G, I, and J are hyperactive responses (see text).

Chart V. This demonstrates variable responses obtained on the same individual with varying amounts of air expired at varying pressures.

Responses A, B, C, and D. An executive 56 years old; obese, weighing 211 lbs., unless instructed to breathe in full lung capacity, invariably presented a poor response at first test, followed

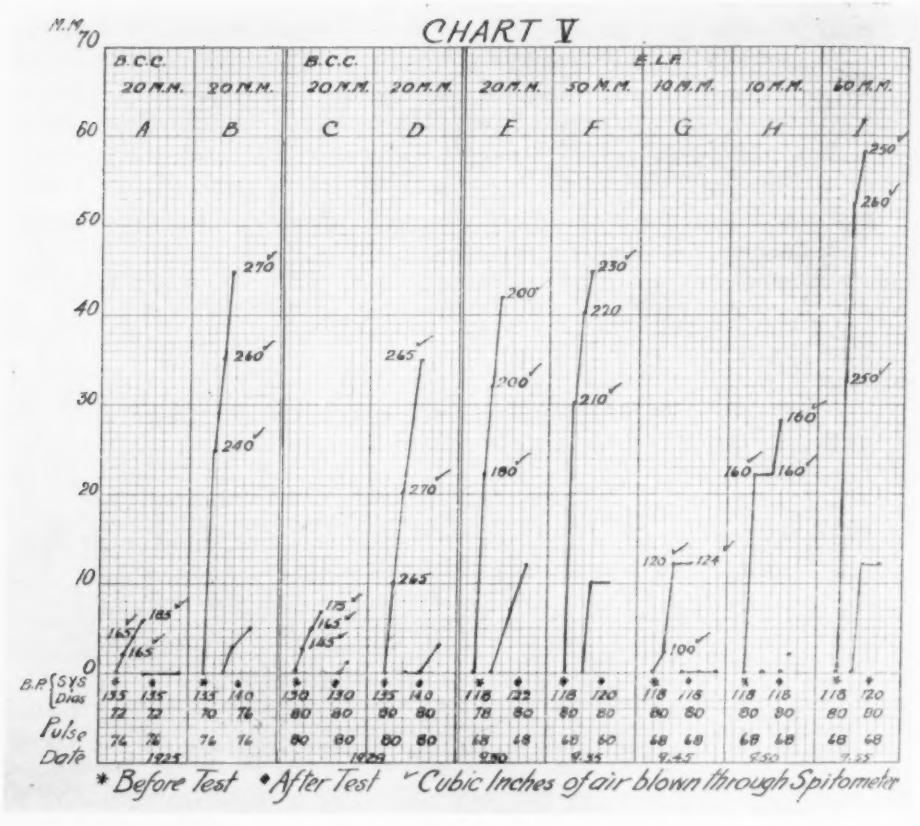

CHART V. Showing responses to cardiorespiratory test when varying amounts of air are expired through spirometer at varying pressure (see text).

in five minutes by a normal response when he was properly cautioned.

Responses E, F, G, H, and I. A normal individual 35 years old, gave a normal response (E) with the proper technique; a slightly higher response (F) when the same amount of air was expired at a pressure of 50 mm. instead of 20 mm. Responses (G and H) are below normal, obtained by expiring varying amounts of air at a pressure of only 10 mm., while (I) was obtained by extreme inspiration and expiration of 260 cubic inches of air at a pressure of 60 mm.

The importance of cautioning the patient to breathe his full vital capacity at a constant pressure of 20 mm. of mercury is manifest from an examination of these marked variations obtained on the same individual. I have experienced no trouble in getting the full co-operation of patients, excepting some women who seem to be unable to breathe deeply.

SUMMARY

1. A modification of the original cardiorespiratory test, devised by Frost, is described, which, on account

of its simplicity, is applicable to routine clinical needs in evaluating cardiac cases.

2. A protocol of 125 cases on whom 160 modified cardiorespiratory tests were performed, is presented.

3. A poor response was obtained in 35 per cent; a fair response in 25 per cent, and a normal response in 40 per cent of the tests.

4. The response to the test corresponded to the clinical evidence of the functional capacity of the heart.

5. Charts of curves plotted from responses obtained in a number of cases observed over several years, are presented, showing graphically the improvement or decline in response to the cardiorespiratory test, corresponding with the clinical picture of the case.

6. A failure of the systolic blood pressure to respond in an increase of at least 20 mm. denotes a weakened heart muscle; while a fall in the curve of the base line denotes a serious dilatation of the heart. The pulse rate, before and after the test has clinical significance.

7. The cardiorespiratory test, when properly applied, is a valuable aid in the diagnosis and study of chronic myocardial insufficiency.

8. The test should be used with caution on cases of marked cardiac dilatation; on cases with a history of angina pectoris, and on cases of hypertension.

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Auricular Paroxysmal Tachycardia Caused by Digitalis

Report of a Case*†

By Arthur F. Heyl, B.S., M.D., *New Rochelle, N. Y.*

While a patient, a man fifty years of age, suffering from congestive heart failure and hypertension, was being digitalized,** he returned to the clinic complaining of attacks of very rapid heart action. He stated that they were abrupt in onset and varied in duration from a few minutes to twelve or more hours. Some of these attacks were observed later in the clinic and thought to be auricular paroxysmal tachycardia. This was confirmed seven weeks after their onset (see EKG no. 148, plate I; compare original EKG no. 123, plate I). Since these attacks were assumed to be spontaneous an attempt was made to prevent them by more complete digitalization, following the suggestion made by Levine and Blatner.[1] The attacks, however, became more frequent with this treatment and since a review of his history indicated that he had not been troubled by palpitation of this type before he first received digitalis, the possibility was evident that the digitalis might be the cause of his attacks. He was then hospitalized for study to prove or disprove this theory.

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†From the New Rochelle Hospital Cardiac Clinic, New Rochelle, N. Y.

**Lederle tablets and Davies, Rose and Company pills of the powdered leaf were the only preparations used with this patient.

History and Physical Examination of the Patient on Admission to the Clinic

The patient, a white male, fifty years of age, hospital clinic number 8739, was seen first on August 1, 1929. His chief complaint, primarily of an asthmatic nature, was shortness of breath during the night and early morning, or with effort. This began about a year previously, following an attack of acute bronchitis.

His past history was essentially negative, except for chronic bronchitis of fifteen years duration and pneumonia at the ages of thirty-seven and forty-five years. He also had a severe attack of "grippe" during the winter of 1928-29.

His family history was irrelevant.

Physical Examination: The patient was a well-nourished, tall, erect male. A most striking over-development was noticed in his full, barrel-shaped chest, with sunken supraclavicular and suprasternal fossae. There was no visible or palpable enlargement of the thyroid gland. The more important physical findings were an emphysematous chest full of sibilant and musical râles and noticeable precordial and left axillary line bulging. There was slight dullness at both bases. Heart sounds both at the base and apex were distant and ill-defined. At the lower left sternocostal angle "apex sounds" were best heard of natural quality. No murmurs could be heard. The apex and pulse rates were equal at 110 and 120, depending upon the position of the patient, reclining or erect, respectively. The blood pressure was systolic,

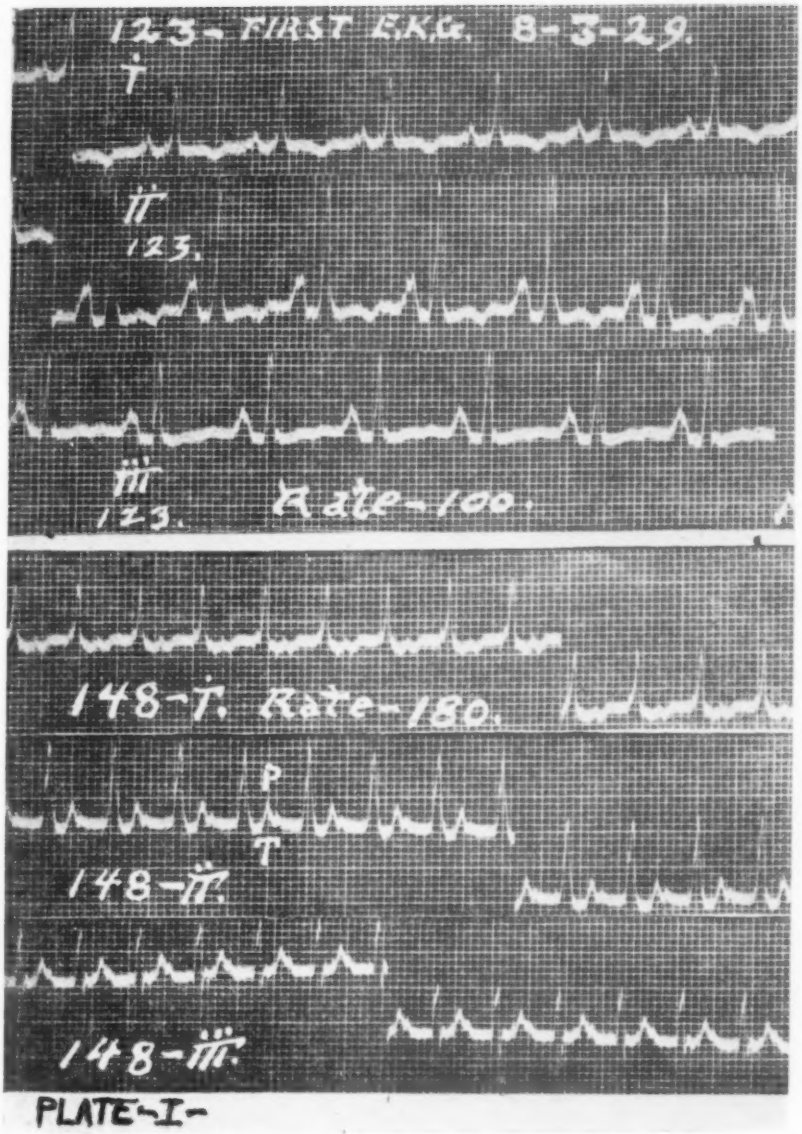

PLATE I. EKG no. 123, initial tracing before administration of digitalis showing left ventricular strain according to Barnes and Whitten.[7] No. 148, rate 180 per minute, one of many recurrent attacks of auricular paroxysmal tachycardia after digitalization, on a maintenance dose of 0.1 gm. a day.

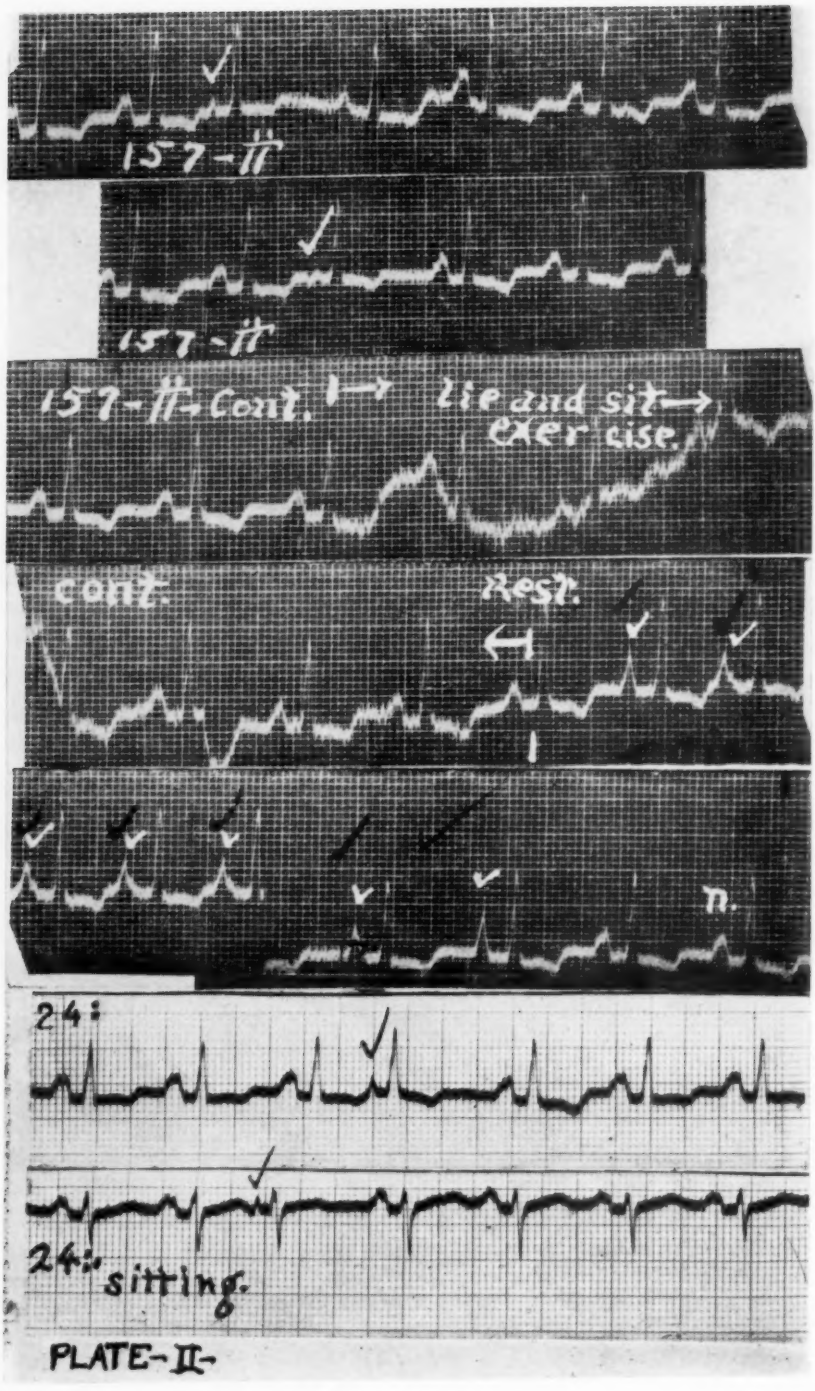

PLATE II. EKG no. 24; leads II and III show rare premature auricular contractions. Note respiratory effect on S-wave. Made ten days after discontinuing digitalis.

170 mm.; and diastolic, 130 mm. of Hg. There was moderate edema of the ankles.

On August 24, 1929, radiographic examination of the chest at 77-inch focal distance revealed an aortic type heart with left ventricular enlargement, right interlobar pleural thickening, emphysema and bronchiectasis (plate XIII).

On Jan. 16, 1930, his blood chemistry was normal, as were also his blood count and urine analysis. His blood Wassermann was negative.

A diagnosis was made of: (1) Chronic heart disease associated with hypertension (pneumonia, chronic bronchitis, emphysema, bronchiectasis, asthma); (2) Greatly enlarged heart; (3) Regular sinus rhythm, occasional premature contractions, sinus tachycardia; (4) Early congestive heart failure.

Although indicating that his attacks occurred only when he was receiving digitalis (EKG no. 183, plate III) the evidence during this hospitalization was not convincing, because he came to the wards after weeks of such medication and no control period free from the drug and attacks of tachycardia had prevailed. He was discharged without medication for the purpose of studying the condition further.

Tachycardia did not occur for three months until the patient on his own responsibility, because of dyspnea, took two grams of digitalis in ten days (0.2 gm. a day). This resulted in an attack of tachycardia which lasted intermittently for four days.

Auricular Paroxysmal Tachycardia Induced by Digitalis Three Times Under Controlled Conditions

After two more months without digitalis, in which period he had no attacks, he was again admitted to the hospital, this time in marked congestive failure. Following a rest of four days in bed without improvement, digitalis was administered. It relieved him of failure but it also induced auricular paroxysmal tachycardia. Two subsequent observations with rest periods of seven days intervening, during which time digitalis was withheld, yielded additional proof that digitalis was the exciting cause of his attacks.

Period One (control EKG no. 243, plate IX). When 1.3 grams of digitalis had been given in twenty hours, the patient complained of palpitation and an auricular paroxysmal tachycardia was revealed (no. 247, plate IX). Digitalization was continued to the full calculated dose of 2.9 grams in three days and then discontinued because of a 2:1 block, with a rapid ventricular rate. The abnormal auricular rate continued (no. 248, plate IX). There were no other toxic symptoms. Three days later, transient "paroxysmal" attacks occurred as the result of ventricular response to each auricular impulse (no. 250, plate IX). Subsequently, the patient's usual sinus tachycardia prevailed, rate of 100 to 110 per minute (no. 251, plate IX and no. 252, plate X).

Period Two. An interval of seven days elapsed without the administration of digitalis. After the patient had received 0.9 gram in twelve hours, he was awakened by tachycardia two and one-half hours following the third dose of 0.3 gram. This occurred at night and no tracing was obtained. Due to an additional and final dose of 0.3 gram four hours later, a 2:1 block with an auricular rate of 220 per minute resulted (nos. 254 and 255, plate X). In the next four days, transient periods occurred, however, when the auricular

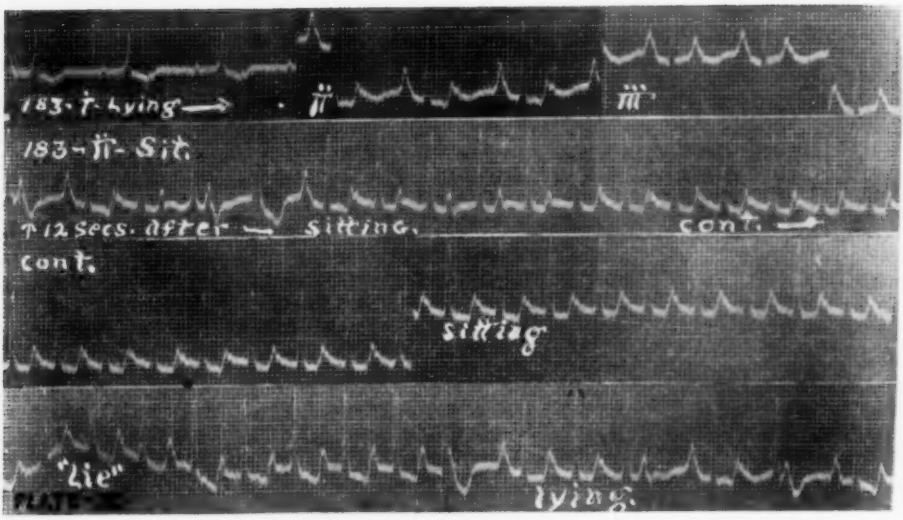

PLATE III. The last three strips are continuous.

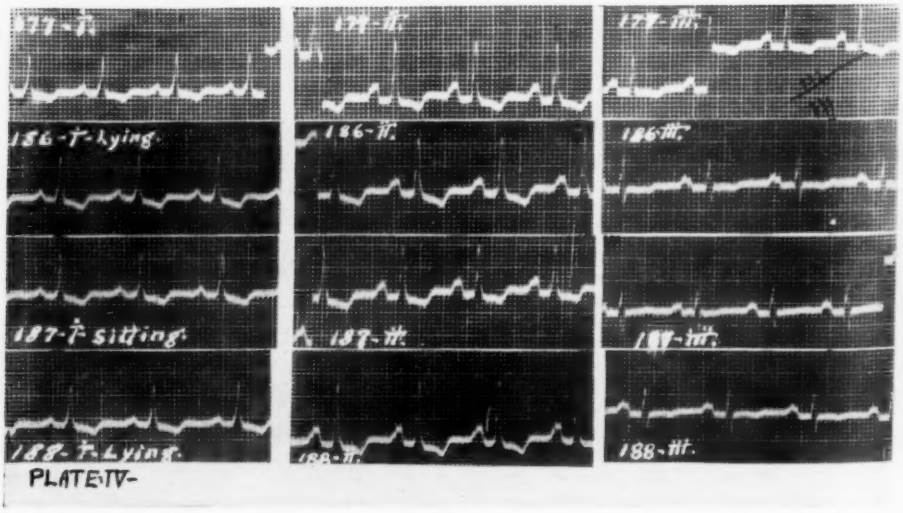

PLATE IV. EKG no. 177, control 5 days prior to 183; nos. 186, 187, 188 taken during 5-day interval after 183 when digitalis had been temporarily discontinued. Compare postural effects in leads III.

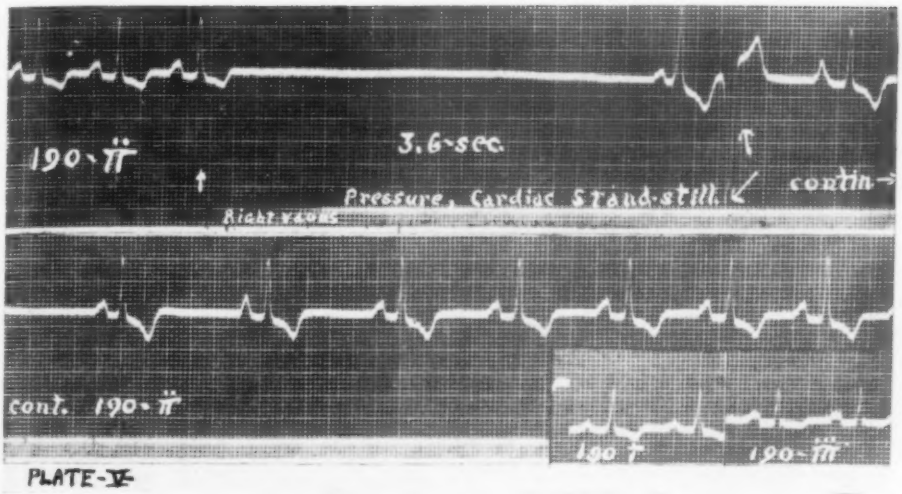

PLATE V. Vagus pressure released at end of "stand-still."

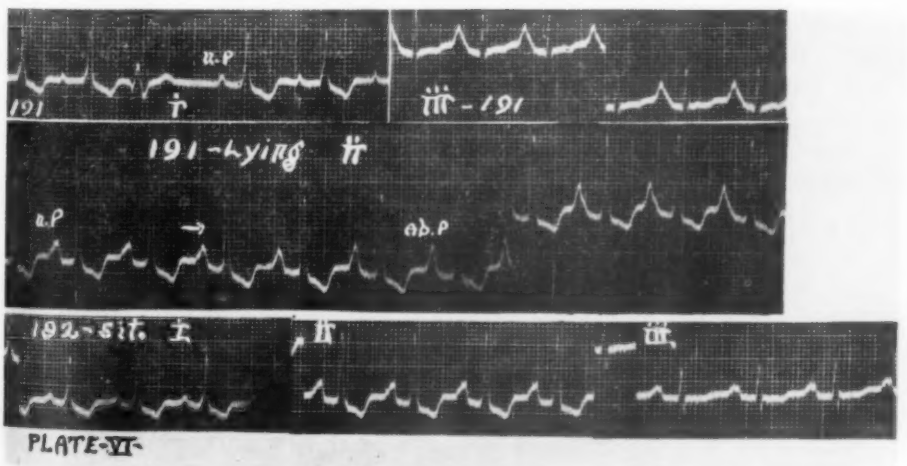

PLATE VI. EKG no. 191, control made 20 hours after no. 190 showing transition in lead II from the "normal" to the ectopic P-waves and an increase in rate. No. 192, control four hours later.

tachycardia was attended by a 1:1 rhythm alternating with block (EKG nos. 256 and 261, plate X). Thereafter, for three days a "normal" rate and rhythm were constant (nos. 262, 263 and 266, plate X).

Period Three. Another week having intervened since the administration of digitalis, 0.9 gm. was given within ten hours. Three hours after two-thirds of this amount had been taken, he had a regular sinus rhythm, with a "normal" rate (no. 267, plate XI). Three and three-quarters hours after the final third (0.3 gm.), the patient complained of tachycardia (no. 268, plate XI). Block again followed for two days (nos. 269, 270, 271 and 273, plate XI) and on the second and third days after digitalis had been discontinued, short periods of palpitation (1:1 rhythm) were again observed by the patient and internes but no electrocardiograms were taken. A "normal" rate with regular sinus rhythm subsequently prevailed (nos. 274; 275, not reproduced; and 276, plate XI, recorded on succeeding days).

Additional Observations

In an attempt to obtain more information relative to the origin of this patient's paroxysmal attacks and the relation to them of digitalis, additional observations and tracings were concurrently made. The effect of vagus stimulation by compression and eye-ball pressure; of exercise; of forced coughing; of holding a deep inspiration; all before and after the administration of digitalis, and the effects of amyl-nitrite inhalation, when no digitalis had been given, were studied.

Vagus Stimulation. At this time, no digitalis having been administered for forty-six days, right vagus compression, left vagus compression, right then left eye-ball pressure, all failed to alter materially the control electrocardiogram (no. 293, plate XII).

During digitalis administration, vagus stimulation gave results which varied apparently with the dosage. Right vagus compression produced cardiac standstill for 3.6 seconds and slowing subsequently to a rate of 40 and 50 per minute, after the patient had received 1.2 gms. of digitalis in divided doses during a period of twenty-five hours. The P-waves were sharpened and the T-waves deepened (no. 190, plate V).

Following an increase in the daily dose of digitalis, vagus pressure failed to produce cardiac standstill. Sharpened P-waves, lengthened P-R intervals and a 2:1 block did result (no. 193, plate VII).

With still another increase in the daily dose, the only result of left vagus compression was expressed by sharpened P-waves. Right vagus compression at first almost completely obliterated the P-waves and reduced the P-R interval from 0.20 to 0.12 second (nodal rhythm; dislocation of pacemaker). The T-waves were not affected as before (no. 198, plate VIII). Space does not permit reproduction of the electrocardiogram but in this same tracing right and left eye-ball pressure separately gave the same results as right vagus (neck) compression.

At another time, two days after 1.2 grams of digitalis had been given in 18 hours, a run of auricular paroxysmal tachycardia was interrupted by right

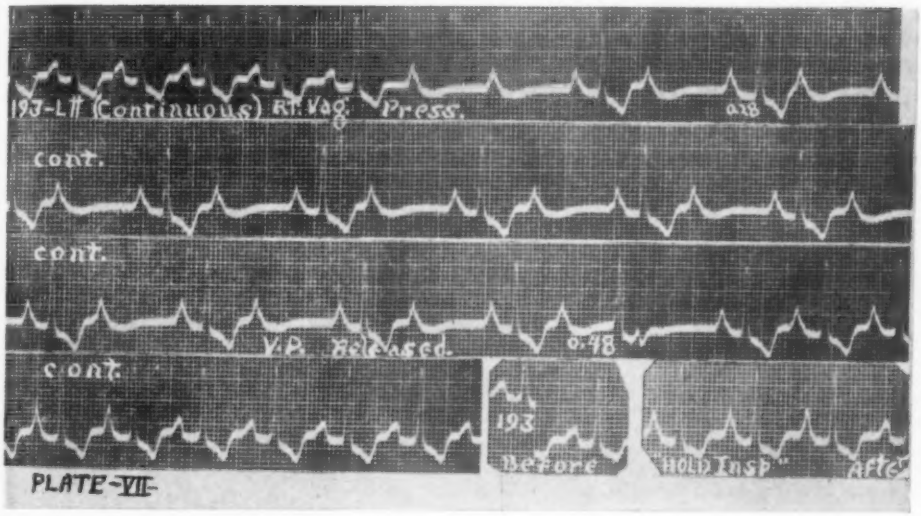

PLATE VII. Note the gradual transition in the last strip from ectopic to normal rhythm following vagus release.

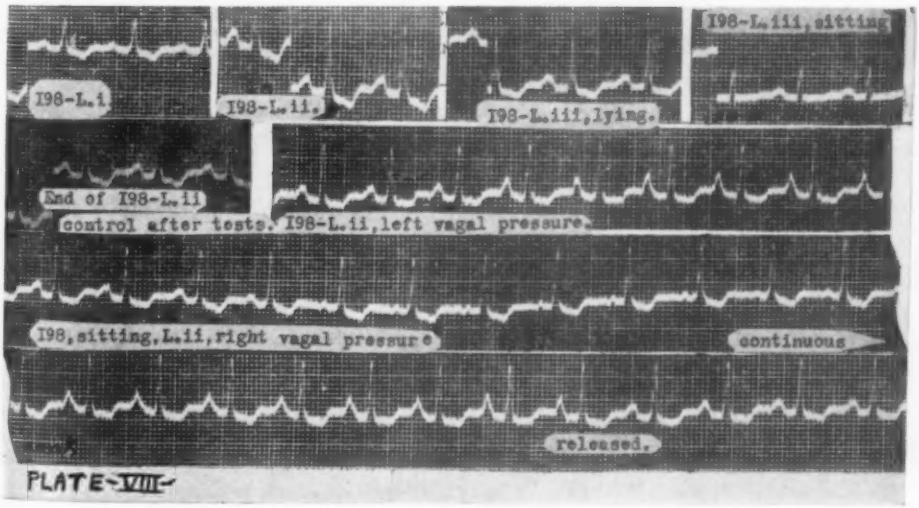

PLATE VIII

vagus pressure and resulted in 5:1, 3:1, 2:1 block (no. 256, plate X).

The only effect of exercise after digitalis had been withheld for forty-six days was to increase the rate from 100 to 110 per minute. After digitalis had been administered, exercise resulted in tall, sharply pointed, ectopic P-waves (no. 157, plate II) like those appearing in his first recorded auricular paroxysmal tachycardia (no. 148, plate I). On another occasion, the effort of sitting up in bed transformed an auricular paroxysmal tachycardia, with a 2:1 a-v block, auricular rate of 180 per minute, to the same type of tachycardia with auricular and ventricular rates of 150 per minute. Reclining again resulted in the original block (no. 183, plate III). These variations were immediately repeated and recorded three times with the same result.

The act of coughing produced the characteristic ectopic P-waves, digitalis previously having been administered (no. 267, plate XI). This effect also was observed when no digitalis had been given.

Holding a deep inspiration induced the ectopic P-waves, following the administration of digitalis (no. 193, plate VII). When digitalis had been withheld for many days, this could not be duplicated.

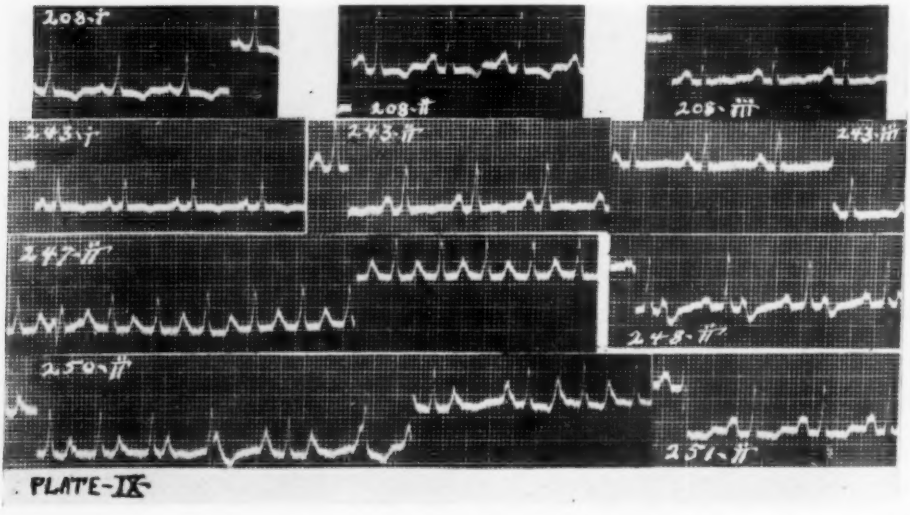

PLATE IX. EKG no. 208, control after 1 month without digitalis. No. 247, rate 150 per minute.

The only effects of amyl-nitrite inhalations six days after digitalis had been discontinued were an increase in the rate from 100 to 120 per minute and slight elevation and sharpening of the P-waves (no. 277, plate XII).

Spontaneous Premature Contractions

Spontaneous auricular premature contractions were recorded in only two of the forty-five tracings from this patient made during more than one year's observation. In the first in-

stance they occurred during digitalis administration (no. 157, lead II, plate II). Nine months later, when no digitalis had been given for ten days, such premature contractions of spontaneous occurence were recorded (no. 24, leads II and III, plate II).

Spontaneous ventricular contractions also were rare (no. 191, lead I, plate VI; no. 247, lead II, plate IX).

Comments

A thorough search of relevant work published since 1912 failed to reveal an instance of auricular paroxysmal tachycardia that was definitely proved to be due to digitalis.

Bastedo[2] has reported a case of paroxysmal (auricular) tachycardia which he considered as produced by digitalis, but the origin of this disturbed cardiac mechanism is not accurately demonstrated since the tracings are polygrams.

Howard[3] presents one case and cites eleven others from the literature of double, auricular and ventricular, tachycardia due to digitalis. He published only one electrocardiogram from his patient in which he found co-existing auricular and ventricular tachycardia with rates of 195 and 160 respectively. The P-waves, as he states, are inverted and therefore "this represents an auricular tachycardia originating in an ectopic focus in the auricle"; but the repetition of such production was not obtained because of the patient's death. It is not possible to state with certainty that the digitalis was the cause.

Luten[4] reported four patients with normal cardiac mechanisms in whom temporary auricular tachycardia developed with atrioventricular dissociation after receiving large amounts of digitalis. No mention is made of their being paroxysmal in type, having their origin in ectopic foci. Reporting an electrocardiogram of his Case 3, he expressed the belief that the rhythm originated in the auricle. "Auricular waves, however, cannot be clearly distinguished, either in this paroxysm or in that part of the record which immediately precedes it."

With the exception of Howard's case none of the eleven others which he reported, including Luten's, apparently conform to the accepted definition of paroxysmal tachycardia, having its origin in an ectopic auricular focus (Lewis[5]), and in none of them do repeated cause and effect observations make it possible to state with certainty that the digitalis induced the attacks.

While in our case a tracing was not obtained showing the actual onset of a usual attack, it was observed clinically to be abrupt and furthermore, the ectopic origin of the P-waves is clearly evident.

A midday control tracing (no. 191, lead II, plate VI), taken by the technician, does show the gradual development of the ectopic P-waves and a progressive shortening of the T-P interval with an increasing rate. This no doubt is the manner in which the paroxysmal attacks originate in this patient and from which the 2:1 block at a higher stage of digitalization results. Further, in the last six complexes of no. 193, plate VII, following the release of right vagal pressure, is shown the gradual relinquishment of such an ectopic rhythm corresponding

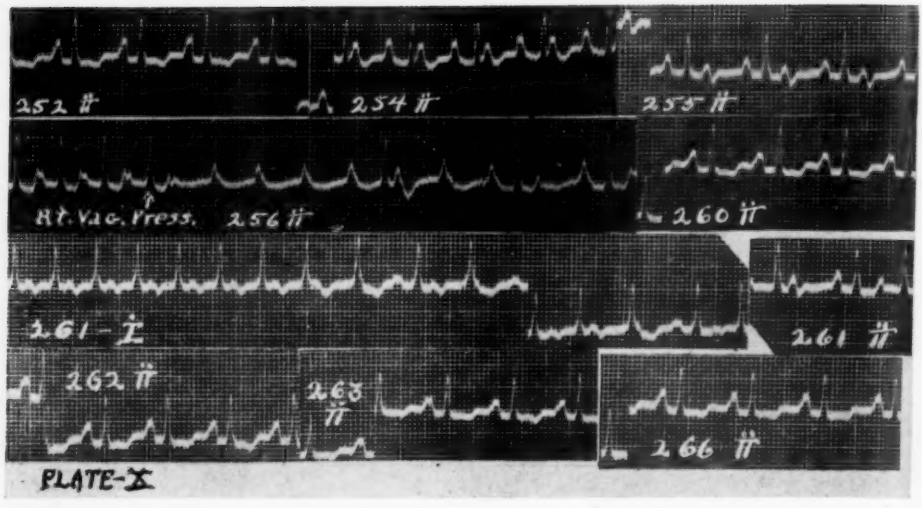

PLATE X. EKG no. 254, auricular rate 220 per minute. No. 255, auricular rate 180 per minute.

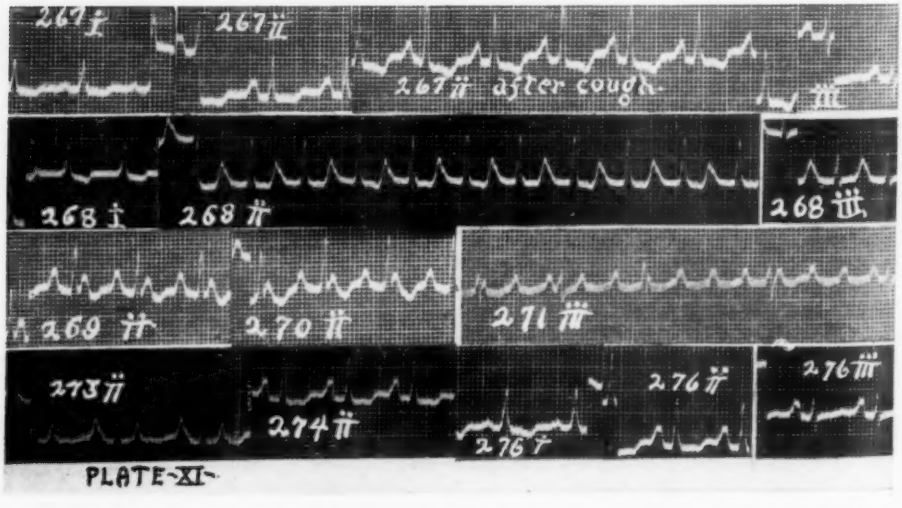

PLATE XI. EKG no. 267, three hours after administration of total of 0.6 gm. digitalis. No. 268, three hours and forty-five minutes after final total administration of 0.9 gm. digitalis.

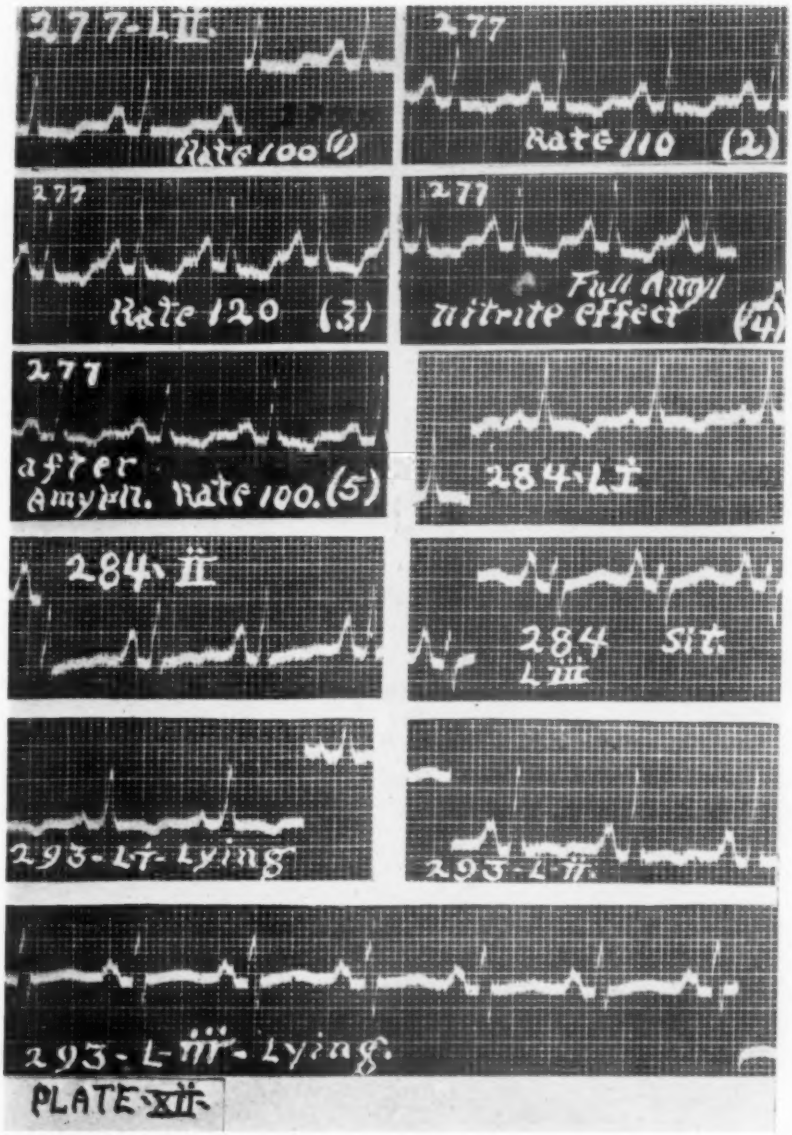

PLATE XII. EKG no. 284, control 26 days after discontinuing digitalis. Note postural and respiratory effect on deviation of electrical axis as compared with No. 293 twenty days later.

to an end phase of his auricular paroxysmal tachycardia.

In electrocardiogram no. 183, plate III, while the auricular paroxysmal tachycardia is present with a 2:1 block, the onset of 1:1 rhythm, the release of block, is nicely shown, occasioned by sitting up in bed. The interruption of such ventricular response, re-establishment of a 2:1 block, is also shown. Such transitions likewise are seen in no. 250, plate IX, without previous effort.

Summary and Conclusions

1. Presented with an adult male, past middle age, having hypertension and congestive heart-failure, it was observed more than nine times that the digitalis, which gave him relief from his symptoms (dyspnea, cough, passive congestion, edema) concomitantly induced auricular paroxysmal tachycardia followed by a 2:1 block, in which the abnormal auricular mechanism prevailed.

2. These abnormal rates and rhythms occurred only as the result of digitalis administration.

3. The writer was unable to find any reference in the literature to such a mechanism due to digitalis so conclusively proven.

4. Various methods, such as effort, deeply held inspirations, forced coughing and amyl-nitrite inhalations were utilized in an attempt to induce the attacks, both in the presence and in the absence of digitalis administration. When no digitalis was being administered they were consistently unsuccessful. Ectopic P-waves like those occurring with his attacks were, however, obtained, but in only one instance, by forced coughing, when he was free from the drug.

5. Various attempts were made to ascertain the effects of vagus stimulation. While he was free from digitalis, external vagus stimulation did not produce any change in the cardiac rate or rhythm nor any electrocardiographic variations. Neither did digitalis alone, even in full dosage, slow the rate as would have been true had it acted directly on the vagus. But the heart at different times, affected by different amounts of digitalis, was rendered susceptible to external vagus stimulation in effects varying from complete cardiac standstill to 5:1, 3:1, 2:1 block, with auricular slowing; to nodal rhythm and no slowing; or to ectopic P-waves and no slowing, depending on the size of the dose. These vagus responses, occurring only in the heart affected by digitalis, correspond to the method of Weil[6] for determining the onset of toxic digitalis effects.

6. Spontaneous premature contractions were exceedingly rare, either of auricular or ventricular origin, in the presence or absence of digitalis administration.

7. No toxic symptoms due to digitalis ever occurred, even with full calculated dosage, except the abnormal mechanisms previously mentioned.

8. The practical significance of these observations is, as others have noted, that graphic methods are especially valuable for diagnosis in patients with sinus tachycardia, particularly in those who need and are receiving digitalis. In this instance even mild doses repeatedly resulted in auricular paroxysmal tachycardia, with or without a

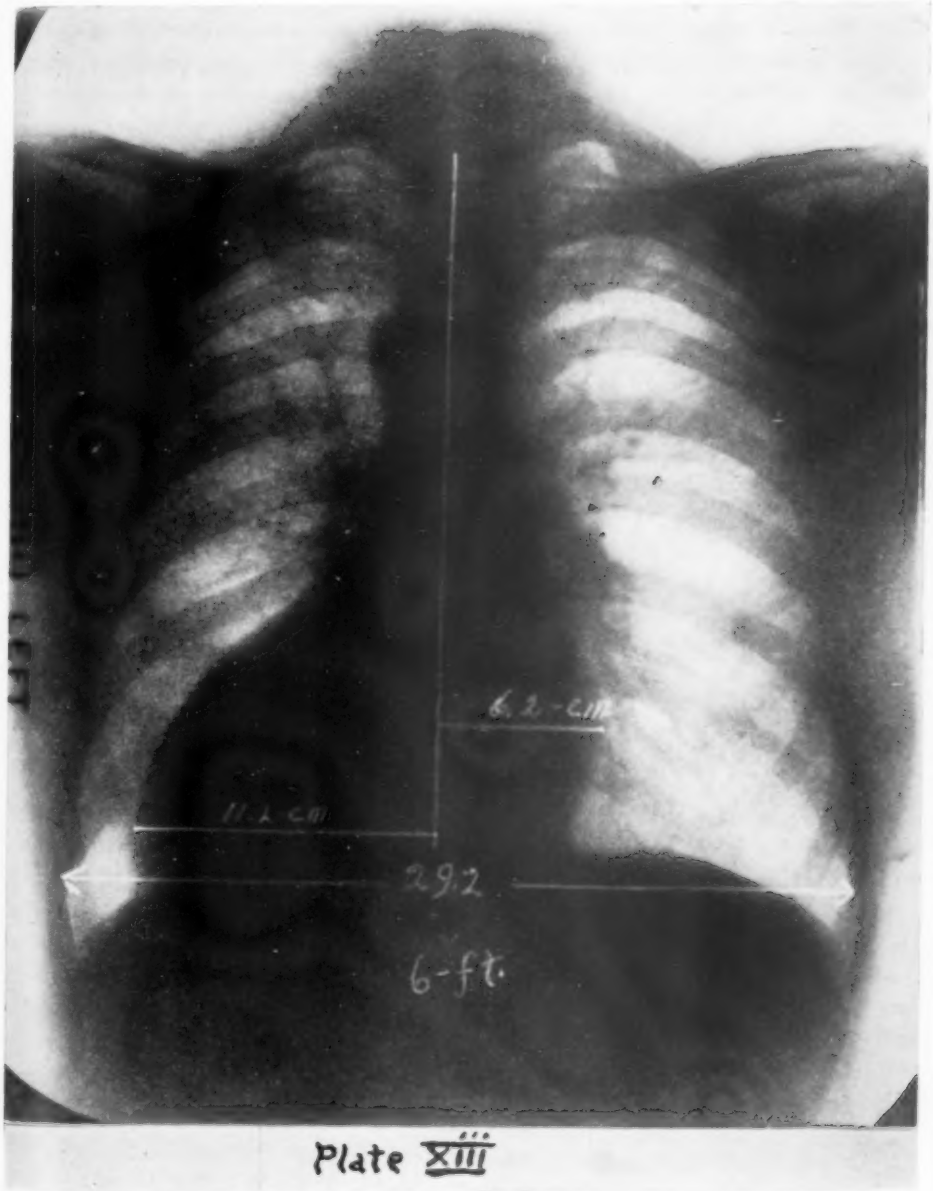

PLATE XIII

2:1 block with a rapid ventricular rate, which without electrocardiographic control, might naturally have led to the futile use of more digitalis and increased toxicity in an effort either to slow the ventricular rate or to prevent the often recurring "clinical" auricular paroxysmal tachycardia.

I am indebted to Dr. Harry Gold of New York for his helpful criticism and suggestions in the preparation of this report.

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Hospital Diets

"A ROUTINE hospital diet should no more be a mysterious affair than a prescription for the pharmacist. If its existence is at all justified, it should be based on the same rational principles that govern all other forms of therapy. A routine diet should have a purpose, a plan, and an objective; it should be based on the laws of nutrition; it should contemplate specific needs in the patient; it should have its indications and its contraindications. Properly employed, the routine hospital diet becomes a convenience to the physician, to the administrative staff, and to the patient."—(From *Clinical Dietetics*, by Harry Gauss, M.S., M.D., F.A.C.P., C. V. Mosby Company.)

Medical and Surgical Problems Associated With Coronary Sclerosis*†

By Arlie R. Barnes, M.D., F.A.C.P., *Rochester, Minn.*

TODAY, beyond doubt, heart disease is the "captain of the men of death." Coronary sclerosis accounts for a large proportion of deaths from heart disease. This might not be so depressing if death from coronary sclerosis came after a long life, but too often the condition claims its victim when he is in his prime and when elsewhere in his body there is no evidence of serious deterioration. There is a distinct need for a review of the common aspects of this disorder, to bring into sharper view its clinical manifestations, and to call attention to the bearing the newer knowledge has on the solution of the many problems which coronary sclerosis presents.

Additions have been made to knowledge of the normal coronary circulation and its pathologic changes. Spalteholz, Gross, Whitten and Campbell have greatly enlarged knowledge of the normal coronary circulation. The distribution of the branches of the right coronary artery to the basal and posterior portion of the left ventricle, described by these writers, has not received the attention it deserves and the result has been failure to appreciate the frequency with which that portion of the left ventricle may be the site of infarction. The work of Whitten disclosed striking variation in the structure of the coronary arteries, depending on whether they are distributed to the right or the left ventricle. Branches of either the right or the left coronary artery, going to the left ventricle, leave the main trunks at right angles, penetrate the myocardium, and when they reach the endocardial surface turn at right angles, ending in a mass of fine arterioles. This has the effect of fixing the main trunks at the points of origin of the penetrating branches. When arteriosclerosis occurs, with resultant lengthening of the vessel, it leads to more or less angulation of the main branches at the points of fixation. This, together with the disproportionate degree of arteriosclerosis that occurs about the mouths of these penetrating branches makes for greater narrowing at these points, and increases the liability to thrombosis of the main branches.

On the other hand, the smaller branches of the right coronary artery, going to the right ventricle, spread out in the same plane as the main divisions from which they arise, and these small branches anastomose freely. This may be one explanation of the almost total

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absence of acute infarction in the right ventricle.

Gross is of the opinion that with advancing years there is increasing impoverishment of the circulation through the right coronary artery as compared with that through the left. Whitten's studies did not support that view, for he wrote: "The portion of the right coronary artery which supplies the left ventricle keeps pace in its vascular development with the left coronary artery in successive decades." Certainly if myocardial infarction can be considered as an index of the inadequacy of the coronary circulation, the overwhelming frequency of its occurrence in the left ventricle indicates failure of the circulation to the left ventricle with advancing years rather than failure of the circulation of the right ventricle.

Comparative anatomy shows that the presence of coronary arteries is associated with the development of a cortical myocardium.[25] In the lower animals the blood circulates in the intratrabecular spaces, through the whole thickness of the cardiac walls. Higher in the animal scale, in reptiles, the inner trabecular part of the myocardium retains a thebesian, sinusoidal circulation while there is a definite coronary circulation to the cortical portion of the wall. There is free communication between these two systems of circulation. In the rabbit "the thebesian, intertrabecular circulation is much reduced, but it retains its connections with the coronary capillary system, and persists as an integral part of the adult myocardial blood supply."[25] It is not surprising, therefore, that Wearn[26] and his coworkers[17] have been able to demonstrate in man communication of the coronary circulation with the chambers of the ventricles, either by way of the thebesian vessels or by means of coronary capillaries communicating directly with the ventricular chambers. Cases of bilateral occlusion of the ostia of the coronary arteries have been reported. The occlusions presumably had occurred gradually, and probably had existed for some time prior to the attack. It is apparent that in such cases the circulation to the heart had to take place by way of the thebesian veins, through their connection with the coronary circulation.

Besides the thebesian circulation, there is a variable degree of pre-capillary anastomosis of the coronary arteries. The coronary arteries are no longer considered to be strictly end arteries. The degree of anastomosis appears to have individual variation, and on this account it may be assumed that some persons are inherently endowed with a coronary circulation which is little able to cope with acute closure of a coronary vessel. The degree of anastomosis tends to increase with advancing age, and that may be one reason why fewer persons more than seventy years of age die of acute coronary occlusion than those of an earlier age. The rate of obliteration of coronary vessels also plays an important part in the extent to which anastomotic channels develop. The studies of Oberhelman and LeCount may be interpreted to mean that in those normal hearts in which there is a negligible amount of anastomosis of the coronary vessels and in which the arteries are essentially end arteries, sudden occlusion of an artery is likely to result in sudden death. In hearts

which are fairly normal, and which possess rich collateral circulation, sudden occlusion of a vessel may be well tolerated, may lead to infarction and subsequently to healing by fibrous replacement. In hearts with slow development of sclerotic narrowing, abundant coronary anastomosis is likely to occur and it is in this group that sudden coronary occlusion is best tolerated. As Benson remarked, "It must be concluded, then, that arteriosclerotic narrowing of the coronary arteries as related to thrombosis of these vessels, is not an unmitigated evil in that it gradually prepares them for the catastrophe that is to come."

The pain observed following acute occlusion of a coronary vessel certainly appears to have a definite pathologic basis. The major difference between this pain and that which is unassociated with coronary occlusion is probably one of degree only. Atheroma of the coronary arteries is the common lesion found in cases of coronary sclerosis, and the pain is considered to be the result of anoxemia[14] due to a blood supply "suddenly insufficient for the needs of the heart muscle for the moment".[10] In certain cases of angina pectoris, sufficient changes in the coronary vessels to account for inadequate blood supply are lacking, and a paroxysmal vasomotor spasm has been postulated to explain these cases. The demonstration by Anrep and Segall that there is vasomotor control of the coronary circulation in the dog might be regarded as supporting the conception of such a mechanism of production of pain. However, the explanation of anginal pain on the basis of paroxysmal vasomotor spasm is at present devoid of any reliable clinical or experimental proof.

Angina pectoris is sometimes observed in cases of pernicious anemia,[33] hyperthyroidism,[12] and paroxysmal tachycardia.[6] There are evidences that in many of these cases the coronary vessels are not the seat of notable disease. It seems highly probable that the factor productive of pain, common to all these mechanisms, is a coronary circulation that is inadequate in the amount of oxygen furnished to the cardiac muscle. It is important to note that angina pectoris arising from any of these conditions is usually overcome by correction of the underlying disturbance. Consequently, the prognosis of angina pectoris, when it arises from these causes, is much better than when the condition has its origin in coronary sclerosis.

Coronary sclerosis affects the main branches of the coronary vessels, diminishing in degree as the smaller branches are approached. Even in cases of malignant hypertension, the arterioles are less affected than are the arterioles elsewhere in the body.[15] The intima is eccentrically thickened, and atheroma is the common lesion. This may lead to gradual occlusion of a large vessel, and subsequent canalization of the occluded portion occurs rather commonly. Thrombi frequently form at the site of ulcerating and calcareous atheromatous plaques, leading to sudden occlusion of the vessel at that point. Syphilis at present is regarded as having little significance in the production of disease of the coronary vessels, except at their origin, where syphilitic aortitis frequently leads to occlusion of the ostia.

The myocardium, in cases of coronary sclerosis, may not exhibit changes, or it may be involved in varying degrees of myocardial necrosis and replacement with fibrous tissue. Diffuse myofibrosis, interspersed with fairly normal muscle fibers, is found in the region supplied by a severely diseased vessel, and probably results from gradual occlusion of the vessel. This process is considered to be due to chronic infarction, and its degree will be influenced by the amount of injury that exists in the vessel, and by the extent of the development of accessory channels of circulation. Hypertrophy of the heart is present in the majority of cases of coronary sclerosis, but this is probably the result of the frequent association of the condition with hypertension. It is not uncommon, however, to encounter hearts of normal size with severe degrees of coronary sclerosis.

Coronary sclerosis and peripheral arteriosclerosis are not necessarily concomitant phenomena. Essentially normal coronary arteries are seen at times in cases of marked peripheral arteriosclerosis. Conversely, the coronary vessels may be markedly sclerosed, while there may be little injury to the peripheral vessels.

Knowledge of the etiologic factors in coronary sclerosis is inadequate. It seems fair to consider that coronary sclerosis is a part of the aging process, and the aphorism that "a man is as old as his arteries" might be changed to state that "a man is frequently as old as his coronary arteries". Coronary sclerosis and coronary occlusion are much more likely to develop in men than in women. A person whose family history contains records of severe vascular disease is predisposed to the development of coronary sclerosis. The ordinary acute infections appear to be unimportant; in fact one is struck by the frequency with which patients who have coronary disease give histories of being remarkably free from episodes of acute infection. Syphilis is a factor in coronary disease in those cases in which syphilitic aortitis produces atresia of the coronary orifices. It is difficult to evaluate the part focal infection plays in the production of disease of the coronary vessels. One gets the impression that patients who have coronary sclerosis have, if anything, less focal infection than the average patient, whereas patients with much focal infection do not seem unduly prone to the development of coronary sclerosis. In my experience, removal of foci of infection in cases of coronary sclerosis has not produced any evident effect on the course of the disease. The occurrence of hypertension accelerates the development of coronary sclerosis, as does diabetes mellitus. Persons with certain constitutional characteristics tend to be victims of coronary sclerosis. Such persons are often powerfully built, and are possessed of great energy and endurance. A life that is strenuous either mentally or physically is frequently the lot of patients who die from disease of the coronary vessels.

The most characteristic symptom of coronary sclerosis is angina pectoris. Heberden's original description[13] of angina pectoris is exact, and little has been added to it since. Heberden wrote as follows:

"They who are afflicted with it are seized while they are walking (more especially if it be up-hill, and soon after eating) with

a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life if it were to increase or continue; but the moment they stand still all this uneasiness vanishes.

"In all other respects the patients are, at the beginning of the disorder perfectly well, and in particular have no shortness of breath, from which it is totally different. The pain is sometimes situated in the upper part, sometimes in the middle, sometimes at the bottom of the os sterni and often more inclined to the left than to the right side. It likewise extends very frequently from the breast to the middle of the arm. The pulse is, at least sometimes, not disturbed by the pain, as I have had opportunities of observing by feeling the pulse during the paroxysm. Males are more liable to this disease, especially such as have passed their fiftieth year. After it has continued for a year or more, it will not cease as instantaneously upon standing still, and it will come on not only when persons are walking, but when they are lying down, especially if they lie on the left side, and oblige them to rise out of their beds. In some inveterate cases it has been brought on by the motion of a horse or carriage and even by swallowing, coughing, going to stool, speaking, or any disturbance of mind.

"Such is the usual appearance of this disease, but some varieties may be met with. Some have been seized while they were standing still or sitting, also upon first waking out of sleep, and the pain sometimes reaches down the right arm as well as the left and even down to the hands, but this is uncommon; in a very few persons the arm has at the same time been numbed and swelled. In one or two persons the pain lasted some hours or even days, but this has happened when the complaint has been of long standing and thoroughly rooted in the constitution; once only, the very first attack continued the whole night."

It is often necessary to make a diagnosis of angina pectoris when cardiac abnormalities are not revealed on general examination or by electrocardiographic or roentgenologic study. It is as if one were called on to identify a man by the shadow he casts or by his silhouette and to do that one must be familiar with every curve, shadow and gesture of the man. To identify a case of angina pectoris without any assistance from any methods of examination demands a most complete mental picture of the syndrome.

I should like to call attention to Heberden's localization of the pain in the sternal region, inasmuch as pains in the lateral portion of the thorax frequently are construed incorrectly as symptoms of disease of the coronary arteries. If pain occurs in the region of the sternum following exertion the presumption is that the case is one of coronary disease, and only the best evidence is sufficient to attribute the pain to any other cause. On the other hand, when the pain is in the left lateral portion of the thoracic wall, the evidence is against a diagnosis of coronary disease and that diagnosis should be made only after the most careful analysis of the symptoms. If the pain is in the left lateral portion of the thoracic wall one must first think of myalgia, intercostal neuritis, herpes zoster, renal colic, diaphragmatic hernia, adherent pericardium, referred pains caused by arthritis of the thoracic portion of the spinal column, lesions of the spinal cord and even the indefinite pains of neurasthenia often associated with cardiac neurosis.

The most characteristic feature of the pain of coronary disease is that it is precipitated by any factor which increases cardiac work and is fairly promptly relieved when the increased demand on the heart ceases. The on-

set of pain following walking is much more convincing in making a diagnosis than it is when it follows the use of the arms, and stooping or bending. The latter exercises bring into play the thoracic muscle, or the spinal column, and lack the value in differential diagnosis of walking, in which many muscles are used. The relation of the pain to changes of weather often identifies it as of rheumatic origin. The details of the manner in which the patient gets relief may be of diagnostic value. One patient, who had his pain in the precordium at night, and who obtained relief by taking warm drinks or by sitting with his back to the fire, proved to have an area of anesthesia in the left lateral portion of the thorax, at the site of his pain, due to advanced hypertrophic arthritis of the thoracic portion of the spinal column.

The brief duration of the pain of coronary disease should be noted. When a patient is subject to repeated attacks of thoracic pain, which last from a half hour to several hours, one must hesitate to make a diagnosis of angina pectoris. A single episode, or at most two episodes, of prolonged precordial pain, may be shown to be associated with one or more attacks of coronary occlusion, but a series of such seizures cannot be so interpreted.

Coronary sclerosis may manifest itself by other symptoms than angina pectoris. Paroxysmal nocturnal dyspnea, with or without pulmonary edema, may be the chief manifestation of coronary sclerosis. However, this syndrome is most frequently observed in those cases in which coronary sclerosis is associated with essential hypertension.

Willius and Brown found that in twenty-six per cent of patients with coronary sclerosis, the disease was manifested by progressive myocardial failure, unassociated with seizures of pain. Scott, likewise, has emphasized the fact that there is a considerable group of patients without anginal pain or hypertension, who run the characteristic course of heart failure which proves, at necropsy, to have its basis in coronary sclerosis. As he stated, these patients often are considered to be suffering from chronic myocarditis, which is an erroneous term. This term is doubly unfortunate, indicating that the pathologic process in the heart is inflammatory and implying that "chronic infection elsewhere in the body is responsible for the myocardial damage,"[22] for neither of which assumptions is there substantiating clinical or experimental evidence.

And finally, all students of this subject are familiar with the fact that coronary sclerosis, even with coronary occlusion and chronic myocardial infarction, is found in persons who have had no clinical signs of heart disease to the time of their death. This group was designated occult coronary disease by Willius, and comprised forty per cent of his cases.

On general examination there may be no objective evidence of heart disease, and as previously mentioned the diagnosis must rest solely on the interpretation of the patient's symptoms. In a considerable proportion of cases a variable degree of cardiac hypertrophy is present, and this usually occurs with associated hypertension. Significant arrhythmia is relatively uncommon. Pulmonary congestion, and

eventually hepatic enlargement, with edema of the legs, occurs in cases which exhibit the syndrome of chronic heart failure.

The roentgenogram of the heart will confirm the presence of hypertrophy when it exists and often reveals calcareous deposits in the aorta. It may also give evidence of widening of the aortic arch, due to torsion and slight ectasia, a finding that is often interpreted erroneously as evidence of aortic aneurysm. Needless to say roentgenographic evidence has only an indirect bearing on the diagnosis of coronary sclerosis.

The greatest confusion exists in the matter of the relation of the electrocardiographic data to the diagnosis of coronary disease. There is a tendency among clinicians to exclude the diagnosis of coronary disease in the absence of significant electrocardiographic changes, and yet Willius[31] has shown that significant T-wave negativity or abnormalities of the QRS complexes were absent in 63.3 per cent of cases of angina pectoris. As far as my experience goes, coronary sclerosis can affect the electrocardiogram in only three ways. First, a characteristic electrocardiographic change usually is observed in the first two or three weeks following acute coronary occlusion, which in many instances is pathognomonic.[2, 5, 21] This is followed by inversion of the T-wave and a peculiar RS-T contour described first by Pardee and designated the coronary T-wave by him. The term coronary T-wave is freely and loosely used, and yet this is the only instance that warrants such a designation. The T-wave negativity may require six months to two years to disappear after infarction. In the second place, coronary sclerosis may affect the function of the auriculoventricular bundle and may lead to varying degrees of heart block. Finally, it may impair conduction in either division of the bundle branches, leading to varying degrees of bundle-branch block with or without inversion of the T-waves. There is yet another condition in which inversion of the T-wave is observed in coronary sclerosis; namely, when that condition is associated with strain, predominantly of one ventricle, usually due to hypertension. Our studies[4] indicate that this change is primarily due to unilateral ventricular strain rather than to coronary sclerosis. Pardee[20] recently described a large Q-wave in lead III of the electrocardiogram, unassociated with right axis deviation, which he considers to be an indication of narrowing of a coronary branch or branches. In sixty-three per cent of the cases studied, this abnormality was not attended by significant changes in the T-wave. If further critical studies confirm this observation, it will become an important electrocardiographic sign indicative of coronary sclerosis.

Finally, it must be emphasized again that by no means is it necessary to have abnormalities of the electrocardiogram before making a diagnosis of coronary sclerosis.

Acute coronary occlusion and myocardial infarction are much more common events than is ordinarily supposed. Studies which Ball and I made of the incidence of myocardial infarction among 1,000 consecutive patients who came to necropsy showed that there was gross myocardial infarction in 4.9

per cent of hearts. In the group of patients in that series who were more than forty years of age, the incidence is 6.8 per cent. Contrary to present ideas, occlusion of the branches of the right coronary artery that go to the left ventricle was found to be common. It is no longer justifiable to speak of the anterior descending branch of the left coronary artery as "the artery of coronary occlusion". As a logical sequence to these observations we found infarction of the posterior basal portion of the left ventricle to be practically as common as infarction in the apical and anterior portion of the left ventricle. The study further emphasized that myocardial infarction is almost entirely confined to the left ventricle.

One cannot enter into a detailed description of the pathologic changes which follow acute coronary occlusion, but certain features are worthy of comment. Death may occur following acute coronary occlusion so quickly that an anatomic infarct may not have time to form. If one wishes to demonstrate the coronary thrombus in these cases "scissors are unsafe instruments for opening the coronary arteries" because of the danger of dislodging the clot. In cases in which infarction has existed a little longer, its presence may be indicated on gross examination only by a region in which muscle softening has taken place and not by changes in the surface coloring of the myocardium. Rupture of the heart occurs in the first three weeks, as a rule. It is the result of the rapid necrosis of the heart muscle which takes place at that time and not ordinarily the result of cardiac aneurysm, which occurs later, due to thinning of the muscle wall, associated with more or less complete replacement of the muscle with fibrous tissue. Many infarcts will be overlooked unless the pathologist incises the left ventricle, parallel with and at a distance of 0.5 to 1 cm. from the posterior interventricular septum. When the muscle beneath a depression in the surface of the left ventricle is sectioned, an underlying chronic infarction often is revealed. Mural thrombi are formed frequently when the infarction extends through to the endocardium, or in certain cases of prolonged and severe myocardial failure. The danger of a portion of this thrombus becoming dislodged and leading to serious or fatal embolism exists chiefly during the first three weeks after the thrombus is formed. Pericarditis occurs in from twelve to fifteen per cent of cases of myocardial infarction; hence the physical signs of this condition may be anticipated in relatively few cases of coronary occlusion. Two or more infarcts of varying ages may be found in the left ventricle, and this often explains clinical facts and varying electrocardiographic changes that otherwise might prove puzzling.

Acute coronary occlusion frequently afflicts patients not previously subject to symptoms of heart disease. Conner and Holt observed that sixty-two per cent of patients with coronary thrombosis did not give histories of antecedent circulatory symptoms. Although our experience, and that of Levine, has been that careful questioning usually will disclose a history of angina pectoris or of other symptoms of heart disease, yet the fact remains that acute coronary occlusion is frequently the first intimation of a cardiac disorder.

The onset of the attack may be signalized by a severe, prolonged attack of substernal or epigastric pain, occasionally by an attack of severe suffocation, and in rare instances it may occur without pain and with little, if any, dyspnea. The pain is distinguished from the ordinary attack of angina pectoris chiefly by its duration. Often large doses of morphine fail to relieve the patient of pain.

The appearance of the patient following acute coronary occlusion is characteristic in the majority of instances. There is an anxious, grayish facies, and the patient is bathed with perspiration. Dyspnea is usually present. Frequently the patient is nauseated, and severe retching and vomiting are seen in some cases. Fever of one to three degrees develops on the second day and persists for several days. The blood pressure usually falls, sometimes to an alarming degree.

On general examination the apical tones are usually somewhat indistinct, and gallop rhythm is often observed. Pulsus alternans is present occasionally. A friction rub can be heard over the precordium in about twelve to fifteen per cent of cases during the first week. Auricular flutter, auricular fibrillation, or tachycardia of auricular or ventricular origin, is sometimes present. At times the infarct extends high enough in the septum to involve the bundle of His, producing complete auriculoventricular block. Willius[30] reported two cases in which Stokes-Adams syndrome resulted from such involvement of the auriculoventricular bundle.

Leukocytes number from 10,000 to 20,000 during the first week. A secondary rise in the leukocyte count may indicate impending myocardial rupture or an additional acute infarction.

The electrocardiographic tracing is characteristically modified, depending on the site of infarction.[5] When infarction occurs in the anterior portion of the left ventricle and apex, in the region usually supplied by the left coronary artery, the earliest electrocardiographic evidence of the fact is a change of level and contour of the S-T or R-T segment in leads I and II and depression of the S-T interval in lead III. The R-T segment in leads I and II, but especially in lead I, is elevated above the iso-electric line. The segment is likely to be convex, dome-shaped, or sloping downward toward the T-wave. Diphasic T-waves, or T-waves of a monophasic type, are the rule in the earliest stages. It is important to note that leads I and III act conversely, so that elevation of the R-T interval in lead I is opposed by depression of the S-T wave in lead III. The changes in lead II are usually seen to be similar to those in lead I in cases of infarction in the anterior portion of the left ventricle. Two or three weeks after acute coronary occlusion the monophasic or diphasic type of T-wave is replaced by frank inversion. The T-waves are likely to be deep, abrupt, or sharply peaked. It is noteworthy that as the T-wave becomes inverted in lead I, the T-wave in lead III remains upright, and becomes exaggerated and sharply peaked. Of particular significance in this later stage is the rounded contour of the R-T interval in lead I or in leads I and II, preceding inversion of the T-wave. At this stage, the R-T interval is approach-

ing or has reached the iso-electric level.

With infarction in the posterior portion of the left ventricle, alone or combined with apical infarction, precisely the opposite set of conditions is seen. In this case, in the early stages, the R-T segment is elevated in leads II and III and depressed in lead I. The same convex, dome-shaped, or sloping R-T segment preceding the T-wave is apparent in leads II and III. In the later stages, the R-T segment in leads II and III tends to return to and eventually reaches the iso-electric level, and the depressed S-T interval in lead I disappears. In this stage, inversion of the T-wave in leads II and III, with a rounded contour of the preceding S-T segment, is observed, whereas the T-wave in lead I is upright, and becomes exaggerated and more sharply peaked. It must be emphasized that if the patient survives for a period varying from six months to two years, evidence of inversion of the T-wave tends completely to disappear and the electrocardiogram returns to normal.

After the first few days, and for a period of possibly three weeks, portions of the mural thrombus formed over the site of infarction may be dislodged, producing embolism in the systemic arteries. If these emboli lodge in cerebral vessels they are likely to cause death. Death may also occur from rupture of the myocardium due to necrosis extending rapidly through the ventricular wall. A considerable number of patients die from congestive heart failure.

Approximately half of the patients survive acute occlusion of a coronary vessel. Some of these continue to have attacks of dyspnea or anginal pain. Many have subsequent attacks of coronary occlusion to which they succumb. A considerable proportion of those who survive are free of trouble in six to eighteen months, and are capable of assuming their usual activities with little, if any, reminder that they have been victims of such a vicious cardiac insult.

The problem of coronary sclerosis and occlusion concerns the surgeon in three main ways. First it is obviously important to recognize or to be able to exclude this condition in cases in which operation is to be performed for any cause. Searching interrogation of the patient for a history of anginal seizures, of prolonged attacks of substernal pain or of prolonged seizures of suffocation must always be carried out, especially if the patient is a man of more than forty years of age. Inversion of the T-waves, not associated with cardiovascular conditions that are capable of producing unilateral ventricular strain nor with increased width of the QRS interval of the electrocardiogram often give a clue to the existence of previous coronary occlusion. The peculiar character of these T-waves has been well described by Pardee.[19] Inverted T-waves in leads II and III are commonly indicative of myocardial infarction, particularly if digitalis has not been given. Obviously, an antecedent history of angina pectoris calls for more lively suspicion that infarction has occurred previously.

If it is recognized that acute myocardial infarction has recently occurred, and the patient is in need of a surgical operation, it is advisable to postpone operation, if possible for three months, for the studies of White

have shown that healing is fairly complete in that length of time. Of course, operation may be undertaken then only if examination demonstrates that cardiac compensation is satisfactory. Under certain urgent conditions it has been found possible successfully to perform a major operation on patients whose acute coronary occlusion occurred as recently as two weeks prior to the operation. However, this is permissible only in emergencies, or in cases in which attacks have been mild, have been accompanied by a minimal amount of shock, and in which there is no evidence of heart failure after the attacks.

The surgeon, and the internist as well, are vitally interested in distinguishing acute coronary occlusion from pathologic conditions in the abdomen, particularly cholelithiasis, perforating peptic ulcer, intestinal obstruction, and acute pancreatitis. This difficulty comes about because acute coronary occlusion may register its pain in the abdomen, whereas, the abdominal lesions enumerated may produce pain in the lower part of the thorax with radiation of pain to the shoulders and even at times to the arms. As Tuohy remarked, collateral and associated evidence is often of more importance than direct evidence and symptoms in arriving at the correct diagnosis. Although the differential diagnosis cannot always be made, yet vivid appreciation that coronary thrombosis can simulate the abdominal conditions named will avoid many embarrassing surgical experiences.

First and foremost, one must be ever mindful of the menace of coronary occlusion among patients who are more than forty years of age, and particularly if the patient is a male. An antecedent history of angina pectoris or paroxysmal nocturnal dyspnea speaks strongly for the possibility of acute epigastric pain having its basis in acute coronary obstruction. The absence of such a history does not allow one to exclude from consideration acute coronary occlusion, however. The presence of essential hypertension, or its preëxistence, predisposes to an attack of coronary occlusion.

The most important step in distinguishing acute conditions in the abdomen from acute coronary obstruction, is the careful taking of the anamnesis. This is particularly true if patients have cholelithiasis or peptic ulcer; in such cases a painstaking review of the history usually will suffice to establish the presence of these conditions. If one remembers that cardiac arrhythmia, muffled apical heart tones, gallop rhythm, friction rub and particularly a rapid drop in blood pressure are symptoms of cardiac insult, the differential diagnosis usually can be made. Willius and Fitzpatrick found that the gallbladder was diseased in twenty-four per cent of cases of coronary sclerosis. When disease of the gallbladder or peptic ulcer exists with acute coronary obstruction, it may be difficult or impossible to arrive at a differential diagnosis.

It can now be said that the electrocardiogram assumes the greatest importance in the differential diagnosis of these conditions. Abnormalities of the electrocardiogram which have been enumerated, appear as early as four hours after acute myocardial infarction, and may retain a highly characteristic

appearance for two to three weeks or longer. It is imperative, therefore, for the internist and surgeon to familiarize themselves with these highly diagnostic changes.

In distinguishing acute coronary obstruction from acute pancreatitis and intestinal obstruction, the electrocardiogram may be of diagnostic value if twenty-four or more hours have elapsed after the onset of the attack. The fecal vomiting, "ladder pattern" seen on inspection of the abdomen, and abdominal rigidity will establish the diagnosis of intestinal obstruction as a rule. As between acute pancreatitis and coronary disease the diagnosis must rest on the occurrence of local tenderness and rigidity in pancreatitis, the history relative to coronary disease, the physical signs of cardiac injury, and possibly on the electrocardiographic changes.

Finally, the surgeon is interested in the problem of the surgical treatment of angina pectoris. Here a plea for conservatism must be made. First of all, operations undertaken on patients with angina pectoris are done at considerable risk. Second, if the surgeon is successful in alleviating pain there is no reliable evidence to indicate that he has modified the serious pathologic process that is the basis for the pain. It is even debatable how much one is justified in depriving the patient of the signal which will warn him that his heart is in distress. At present we are inclined to reserve attempts at surgical relief to patients who fail to obtain reasonable help from the combined use of xanthine derivatives and adherence to a strict regimen. If this treatment fails, the surgical procedure of choice is the conservative method, consisting of paravertebral injection with alcohol of the upper five thoracic nerves.

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Pulmonary Infections by the Friedländer's Bacillus (Bacillus Mucosus Capsulatus)*†

By Frank Fremmel, M.D., Karl J. Henrichsen, M.D., and Henry C. Sweany, M.D., *Chicago, Ill.*

SINCE Weichselbaum's[1] work in 1886, it has been known that the micro-organism described by Friedländer[2] in 1882-3, can cause pulmonary disease *sui generis*. Friedländer's conception, however, that it was the cause of all pneumonias, was disproved by Fränkel just prior to Weichselbaum's report. On the other hand, Fränkel considered the micro-organism only a secondary invader and not the primary cause of any pneumonia—a conception that was also partially erroneous, and one that divided medical opinion for many years.

Sisson and Thompson[3] emphasized this and pointed out that Osler as late as 1912 expressed Fränkel's view in his text book on medicine. Gradually, however, reports have accumulated to show the true status of the parasite, viz: that from one to eight per cent of pneumonias are caused by it. The work of Weichselbaum has, therefore, been confirmed by a large number of authors, the most important of whom are Étienne,[4] 1895; Comba,[5] 1896; Smith,[6] 1897; Thiroloix,[7] 1897; Howard,[8] 1898; Beco,[9] 1899; Moiseyeff,[10] 1900; Brinckerhoff and Thompson,[11] 1901; Kakawa,[12] 1904; Stühlern,[13] 1904; Apelt,[14] 1908; Brissaud,[15] 1912; Gouget and Moreau,[16] 1912; Mosny and Pruvost,[17] 1913, and Zander,[18] 1928.

Some of the authors tend to dissociate these infections from other lung infections and to establish them as separate disease entities. Especially is this true of the chronic form.

The unusual features of these infections are a definite and specific bacteriology, a peculiar clinical course with sudden death and extremely high mortality, and a characteristic gross and microscopic pathology.

The clinical course of the disease is not always regular, but most authors speak of a sudden onset, usually without a chill, no herpes labialis, death coming suddenly between two to five days, usually before the third day. Occasionally, however, the process may resemble closely a lobar pneumonia except that it usually terminates more suddenly. The physical findings are given by Weill and associates[19] as those of engorgement rather than hepatization, even in the advanced process. There are signs of some dullness, with suppression of breath sounds, and an absence of subcrepitant râles. This is perhaps due to the large numbers of

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encapsulated bacilli that fill the alveoli and finer bronchi with a sticky mucoid material, preventing the passage of air.

The mortality figures differ slightly, but with the exception of Zander[18] who reported an epidemic of the disease where the mortality was thirty-five per cent, the mortality figures are high. Netter, quoted by Étienne,[4] Brissaud,[15] Mosny and Pruvost,[17] Gouget and Moreau,[16] Kakawa,[12] and others, stated that the disease is nearly always fatal. Lord,[20] in 1915, said that no authentic case had ever recovered. There are sporadic reports, however, of patients of varying grades of chronicity. Recently Belk,[21] Westermark,[22] Collins and Kornblum[23] and others have reported such cases.

Zander's series, mentioned above, seems to be unusual in that it assumed epidemic proportions, yet had a much lower mortality than is reported for the sporadic cases, and perhaps is more truly representative because it is a series nearly as large as all others reported, put together. It shows the effect of such an infection on an average group of people (soldiers in a prison camp), while Friedländer's infection is usually reported as occurring, most frequently in people of low vitality, quite commonly in alcoholics around fifty years of age. It is possible that many recoveries are never reported or even suspected as such infections.

The roentgenologic findings are rather meager. Weill thought that it differs from pneumonia in having a less tendency to show triangles, and Collins and Kornblum,[23] in being less dense, more scattered and nearer the periphery of the lung in location.

The bacteriology is relatively constant. The causal organism is a large round ended, gram negative, heavily encapsulated bacillus. It has since been placed in a distinct group of encapsulated micro-organisms. Although it resembles the organisms found in rhinoscleroma and ozena, and also other members of the group, it is quite different in pathogenicity. Culturally, it resembles the colon-aerogenes group, but differs in its heavy capsules and in a few incidental reactions. Some authors suggest an origin from this group, but such ideas have not met with general acceptance. Notwithstanding the fact that strains of these micro-organisms have a general similarity, there are slight variations that may very well account for some of the clinical and pathological differences.

The pathological changes are the most distinctive and will, when taken together with other findings, differentiate this condition from other lung affections. Moiseyeff,[10] Kakawa[12] and Brissaud[15] emphasized its individuality. While Sisson and Thompson[3] stated that no feature is pathognomic, yet when all are taken together it is quite easy to make a diagnosis. The most important pathologic findings are the pseudolobar consolidation due to a dense confluence of bronchopneumonic foci (rarely true lobar); the absence of a red stage of hepatization; the presence of a mucoid exudate on the cut surface giving it a glazed appearance, the scarcity and irregularity of red cells and fibrin within the alveoli, and the presence of a moderate number of monocytes containing large numbers of the encapsulated bacilli. Hyperemia and thrombosis of the alveolar vessels lead up to a necrosis and ab-

scess formation if the patient survives sufficiently long.

As mentioned above, various reports have been made recently on the chronic forms of this infection with certain variations from the acute stages. This is true of two or three of Belk's patients, and one each reported by Westermarck and Berglung, and Sweany, Stadnichenko and Henrichsen.[24] All of these cases resembled chronic tuberculosis in many respects and likewise three cases reported by Stengel, Collins and Kornblum,[23] all of whom recovered.

It is our purpose to report the history of an extremely acute type of infection with this organism, and contrast that with a chronic type pointing out the transitions from the one to the other.

As the chronic form has been reported previously, only the essential features will be given here for comparison. The other patient, taken from the private practice of one of us (F. F.), had a fulminating type of infection lasting only twenty-six hours, the shortest course on record that we have been able to find.

Case Report

Patient, A. S. Family history: Negative.

Personal history: Age, 49 years; occupation, editor; rubeola and mumps in childhood; herniotomy when 18 years old; used spirituous liquors and tobacco liberally. No record of venereal disease.

Present illness: Began about 6 P. M. with headache and chilliness, and very slight cough. When first seen at 11:30 P. M., the night of onset, the patient was reclining in a Morris chair and appeared quite at ease; however, it was noted that his face was congested and the vermilion borders of his lips were somewhat cyanotic; respirations were 28; temperature, 102.6°F., and pulse, 106. Coughing brought up a sanguineous mucous expectorate. The left pupil reacted sluggishly to light. A coarse tremor of the hands and exaggerated reflexes were also discovered. Neurological examination was otherwise negative. The abdomen was soft; no enlargements and no pain or tenderness present. His mind was clouded; he failed to clearly recognize his friends; restlessness; pronounced orthopnea and decided cyanosis. Respirations were rapid and difficult, and soon developed the Cheyne-Stokes type. Radial pulse was absent and heart tones distant and too rapid for accurate counting. In an apparent effort to raise sputum, his strength failed, so that his heart tones soon disappeared. The blood pressure was 128/84 on first examination; 106/68 the next morning; and 100/70 at 2 P. M. Examination of the blood showed hemoglobin, 70 per cent; coagulation time, 1 min. 30 sec.; erythrocytes, 3,900,000; leukocytes, 2,400; small lymphocytes, and large lymphocytes, 92 per cent; transitionals, 3 per cent, polynuclears, 5 per cent. Urine examination: Sp. gravity, 1,020; highly colored; albumen, 2 plus; few r.b.c., and granular casts were present.

Examination of the chest showed lagging of left side, increased tactile fremitus at left base, bronchial breathing and bronchophony with scattered subcrepitant râles in the same area. A soft systolic blow was heard over the apex. A diagnosis of left lower lobe pneumonia was made.

A decided change for the worse was manifested the following morning. The patient's face was blanched, his hands and feet were cold, and he was perspiring freely. The respirations were shallow, rate 42, and the pulse had lost fullness and had increased to 128 in rapidity. The patient was orthopneic and was in a state of circulatory and respiratory failure. His temperature was 98.°F., and rose to 101.6°F. at 2 P.M. The patient was bringing up with difficulty copious amounts of bloody mucoid sputum.

About 8 P. M. nurse reported the patient had a sinking spell. Examination showed patient in a state of collapse and he expired at 8:55 P. M., about 26 hours after onset.

The pathological findings were as follows: The body was that of a robust appearing male about fifty years of age; height 5 ft.

9½ in.; weight about 200 lbs. There was moderate lividity over face and dependent portions of the body. There were many small pigmented scars over both shins, and a right inguinal scar three inches long. Following a full ventral incision there appeared a normal peritoneum and contents. The right pleura was normal; the left pleural cavity contained about 50 cc. of a purulent fluid and thick gray plaques of fibrin over the left lower lobe and the contiguous parietal pleura. On removal, the right lung was found collapsed and, with the exception of coal pigment in the lymph depots, appeared normal externally and on the cut surface. The left upper lobe was the same, but the lower lobe was completely consolidated and covered with a grayish membrane 2 mm. in thickness beneath which was reddish lustreless pleura. The cut surface was completely consolidated and presented a glossy, slimy surface. The secondary lobules varied from a gray to a pale red-gray, giving the surface a slight mottled appearance.

The only other gross findings worthy of mention were a moderate number of atheromatous plaques on the aortic arch, and an atrophic spleen, both of which suggested but, in the absence of other evidence, did not prove the presence of lues.

The microscopic examination of the lungs revealed a rather uniform granular edema containing scattering monocytes laden with encapsulated bacilli. Very few polymorphs were present. The fibrin was irregularly placed both in the lobules and in the alveoli. It appeared to form a "drift" along the alveolar walls. The alveolar walls were thickened and the capillaries were dilated with red blood cells. Occasionally there was a ruptured capillary with a few free red blood cells in the alveoli.

The patient reported by Sweany, Stadnichenko and Henrichsen,[24] had the following points of difference from the one just given: The course was twenty-eight months and simulated, but varied slightly from, pulmonary tuberculosis. The temperature was irregular, the appearance of the patient was more that of a septic than a tuberculous process. The skin was clear, anemic, and did not possess the usual yellowish tint of tuberculosis. The patient's strength persisted till the day of death. He was able to wash his own teeth and face on that day. Pathologically, the difference was marked. Grossly the lesions began in the right upper, eventually spread by a succession of lesions to the left upper, then downward to both bases. Each lesion seemed to pass through the same evolutionary changes from a focal bronchopneumonia to a walled cavity.

At first there was a local edema, dilatation of the capillaries, with scattering monocytes, in which a few encapsulated bacilli could be found. There were not the large numbers of bacilli that were found in the early lesions of the acute case. Following this "red stage" the lesions extended and the "gray stage" began to show in the centre, while the "red stage" persisted around the border. There was a more marked dilatation of the capillaries with an occasional rupture, an invasion of lymphocytes and plasma cells in and along the alveolar walls, and finally the "resolution" corresponded to an infiltration of polymorphs in the walls of the alveoli, a thrombosis of the capillaries, with a consequent necrosis, abscess formation and excavation. As the cavities enlarged, there was a true metaplasia to squamous epithelium over the walls. Most of the other features resembled the acute case. Between these two, all types are possible, depending on the size of the initial lesion and the duration; a true lobar, a confluent lobular, true lobular, and pseudolobular in series, may result.

Discussion

One of the striking features of this case was the rapid evolution and apparent utter hopelessness of treatment. This is in harmony with the majority of reports. Although the cases reported by Zander[18] and the scattering ones in which the disease becomes chronic, will be encouragement for further effort, there appears to be some unknown toxic substance that overwhelms the patient. This assumption is well supported because only the left lower lobe was involved, yet a profound agranulocytosis of the blood developed, with collapse and death within twenty-six hours from the initial chill. It seems reasonable to suppose that a soluble toxin may be responsible for the sudden prostration. If such should be found, there

Fig. 1. Antero-posterior sagittal section through the middle of the left lung. Huge pneumonic focus in the middle of the lower lobe, extending out to the pleura except at the extreme margins. While this is almost lobar in extent, it is essentially the same type of lesion that begins as a smaller focus.

is no disease in which there is greater need for an antiserum so far as the individual patient is concerned.

Perhaps the greatest need in these infections is promptness in diagnosis. This dictum is true of any disease but, here, from a prognostic standpoint it is especially valuable. The diagnosis can be made only by finding the encapsulated bacilli inside of monocytes in the sputum, the cellular content of which is predominantly monocytic. This, with vague physical signs of pulmonary engorgement, accompanied by a cyanosis and physical state out of proportion to the physical and x-ray

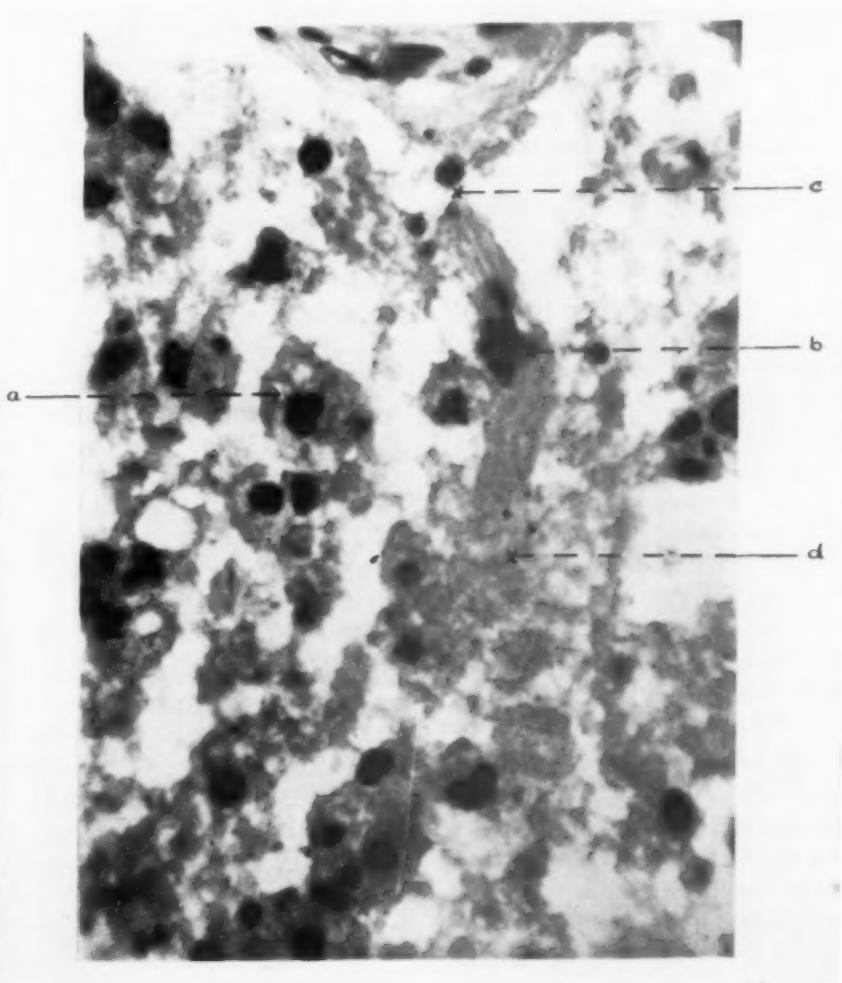

FIG. 2. High power photomicrograph of the pneumonic process, showing a monocyte (a) containing encapsulated bacilli; alveolar wall (b) with necrosis and rupture at 'c' and 'd'. X1090.

findings, establishes the diagnosis. Subsequently, the x-ray shadows become more pronounced (but never like a lobar pneumonia). Bacilli may be found in the blood. It must be differentiated from croupous, broncho-, and (rarely) lobar pneumonia. Although our acute case was a lobar pneumonia, few authors report typical lobar types. Most of them (Brissaud,[15] Kakawa,[12] Moiseyeff,[10] Étienne,[4] and others) consider the most common type a "pseudolobar" or irregularly confluent bronchopneumonia with a less common true bronchopneumonia. Only a few (Brissaud and Kakawa) make mention of a true lobar pneumonia. The differentiation from these conditions must be based on the sputum findings together with the vague and almost negligible physical signs (râles in particular) in an extremely ill patient. The explanation of the paucity of râles is no doubt found in the extreme viscosity of the exudate. This, in turn, is due to the mucoid capsules of the bacilli.

As to the chronic types, they must be differentiated from pulmonary abscesses and gangrene, unresolved pneumonias, particularly that of influenza, and pulmonary tuberculosis. From the common abscess it may be differentiated by the characteristic sputum with the bacilli and the relatively slight odor. From influenza by the sputum with the characteristic bacilli, and from tuberculosis on the bacteriological findings and, according to Kornblum,[23] by the thin walls of the cavities as shown on the x-ray. Our chronic case was indistinguishable, however, from some types of ulcerative pulmonary tuberculosis.

Summary

An extremely acute type of Friedländer's bacillus infection is reported, the patient living only twenty-six hours from onset to death. The clinical findings corresponded to those usually reported for the disease. The pathological findings were those of an uncommon lobar pneumonia instead of a confluent b r o n c h o pneumonia, or a "pseudo" lobar pneumonia. The disease began by a rapid growth of the encapsulated micro-organisms in the alveoli and smaller bronchi, causing an exudate rich in edema fluid containing scattering monocytes, irregularly placed fibrin, with an occasional hemorrhage into the alveoli. This aspect has only a gray to yellow-gray appearance grossly, and accounts for the infrequent appearance of the red stage of hepatization. Death occurred before the other stages could develop. Ordinarily the evolution of the lesion from the "red" stage is characterized by an infiltration of polymorphs along the alveolar walls, forming in crescents along the plugs composed of bacilli, monocytes, and varying amounts of fibrin. Later an invasion of the alveolar wall results in a huge dilatation of the alveolar capillaries, followed by thrombosis, necrosis, and abscess formation. The last two processes correspond to the stage of resolution of pneumococcus pneumonia. The dilation of the alveolar capillaries, with an occasional rupture into the alveoli, give to the lesions a mottled dark red appearance that is occasionally present.

If the patient survives this stage, a gradual change to a chronic form ensues, with a substitution of lymphocytes and plasma cells for the poly-

morphs and an invasion of the older lesions with fibroblasts, connective tissue, and a true metaplasia forming squamous or cuboidal epithelial cells over the cavity walls. When the disease is progressive, there is a continuous sequence of lesions that begins in small bronchopneumonic foci and passes through the various stages of evolution described above, usually extending downward towards the base until death.

The bacteriologic studies included in this report have been made by Miss Asya Stadnichenko, for which we wish to express our gratitude.

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The Phobia of High Blood Pressure

"AT present, the general public, at least in the large cities, is entirely too well acquainted for its own good with the dangers of arterial hypertension. Almost everyone knows some unfortunate who had high blood pressure and died suddenly in the street, or is now paralyzed in half his body. Or when he tells his solicitous friends that he has been discovered to have high blood pressure, they will fill in the gaps in his knowledge of the dangers of the disease. Fortunately, by now the blood pressure has been measured long enough for one to have communicative friends who have had the dreaded high blood pressure for many years and 'never been hurt by it'. It is very common nowadays for one who has always felt well to learn as a result of an insurance or periodic examination or a visit to the doctor for some trivial complaint that he or she has high blood pressure. Then, often enough, the peace of mind of the patient is gone, symptoms make their appearance, and there start the troubles of the patient and, even more, of the family . . . . . At the outset, it is to be emphasized that *many individuals with essential hypertension not only need no treatment whatsoever, but are much better off without it.* Many persons with asymptomatic hypertension would have been more fortunate if they had never learned of their hypertension."—(From *Hypertension and Nephritis* by ARTHUR M. FISHBERG: Second Edition, 1931. Lea and Febiger, Philadelphia.)

Syphilis of the Lung*†

Report of a Case with Autopsy Findings

By Harold Comonfort Denman, M.D., F.A.C.P., *Brooklyn, N. Y.*

Flockemann,[1] whose comprehensive review of the literature of pulmonary syphilis was published in 1898, stated that "the existence of pulmonary syphilis in the adult has not been proved to be very probable." This statement has been doubted by other writers on the subject. Rössle[2] believed that the frequency of pulmonary lues in the adult is under-estimated and reported twenty-five cases which he himself had observed. Allison[3] stated that "Syphilis of the lung in the adult is unquestionably rarely encountered. However it is not so rare as pathologists would lead us to believe, and conversely, not nearly so common as some clinicians and roentgenologists seem to think." This uncertainty, the difficulty of diagnosis and the paucity of characteristic findings in pulmonary syphilis have prompted the report of the following case.

W.G., a laborer, aged 42 years, was admitted to the medical service of Kings County Hospital, November 9, 1930, because of a hemorrhage from the mouth. The following history was obtained from the patient: Two months before admission, he had noticed a persistent cough productive of a small amount of sputum. This cough was never excessive nor painful. The sputum was in small amount, white and never blood tinged. He noticed also a slight but increasing weakness during this period, which did not interfere with his usual work. The hemorrhage which was the cause of his admission to the hospital, occurred suddenly, without cough, pain or unusual exertion, and was described by the patient as a gush into the mouth of blood, bright red in color and slightly frothy. He was unable to state the amount. Associated symptoms were lacking; there were no chills, fever, no sweats, dyspnea, palpitation or precordial pain. He had a fair appetite, no nausea, no vomiting, bowels regular, not medicated. He slept well, and suffered no discomfort from a nocturia, twice nightly, except the inconvenience; and although poorly nourished, had noticed no appreciable recent loss of weight.

The *family history* shows that his father died of erysipelas, age not known to the patient. His mother, two sisters and one brother are alive and well; no history of tuberculosis, malignancy or congenital disease is present.

Previous personal history: During childhood the patient had measles. During adult life the only disease was acute rheumatic fever at the age of 23 years. While serving in the World War he was informed that he had heart disease, but was not discharged for incapacity. His habits were regular, drinking very little alcohol, no coffee, tea freely, but not to excess.

Physical examination revealed a slight, small framed, poorly nourished, pale, adult male about five feet five inches tall, complaining of weakness and a recent hemorrhage from the mouth. Head, eyes, nose,

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†From the Medical Service, Kings County Hospital, Brooklyn, N. Y.

and ears were negative. There were no glandular enlargements in the neck or axilla. Chest examination showed a narrow, asthenic type, with markedly diminished expansion of the entire left side. Tactile fremitus was present anteriorly, to a less extent below the third rib, than over the corresponding area on the right side. This tactile fremitus was increased in the left axilla and absent over the left base posteriorly below the fifth dorsal spine and laterally to the scapula angle. The supra- and infraclavicular fossae were deeper on the left side. There was no dilatation of the superficial veins of the chest. Percussion revealed the presence of a solid body in the third left interspace between the mid-clavicular line and the sternum. Between the mid-clavicular line and the mid-axillary line from the third to the seventh interspaces a hyperresonant note was present. Flatness was present posteriorly below the level of the fifth dorsal spine and laterally to the scapular angle. On auscultation the breath sounds were diminished anteriorly and in the axilla, and increased at the level of the third space anteriorly with occasional loud "bubbling" râles over this area, and absent over the flat base posteriorly. The voice sounds were diminished slightly anteriorly and not transmitted at the base posteriorly. There were fine crepitant râles over the left apex only after an expiratory cough, none at the base. The right side gave the physical signs of compensatory breathing.

The cardiac apex impulse was visible and palpable 8 cm. from the mid-line in the fifth interspace; the rate was 100, normal sinus rhythm. There were noticeable carotid and subclavian pulsations. The left border was percussed 8.5 cm. in the fifth interspace; the right border, 4 cm. to the right in the fourth interspace, and the arch, 6.5 cm. at the level of the second interspace. The first cardiac sound was entirely replaced at the apex and base by a harsh systolic murmur, followed by a short, ringing, greatly accentuated second sound. The radial pulses were equal and regular. The abdomen showed no localized areas of tenderness, no palpable masses and no herniae. The inguinal glands were palpable and hard. Rectal examination revealed a normal prostate. The extremities were negative, and reflexes normal. The blood pressure in the right arm was 110/60; in the left, 114/70.

The possibilities in this patient were: abscess of the lung, aneurysm of the aorta, tuberculosis, gumma of the lung, malignancy. Although there was a slight effusion in the left pleural cavity on admission, it was believed to be due to, rather than the cause of, the localized findings. (Figure 1.) Seventeen days after admission the patient showed signs of a rapidly formed, large effusion in the left pleural cavity and a roentgen picture at this time proved the presence of an effusion, which on aspiration was bloody. (Figure 2.)

During the patient's stay in the hospital, his main complaint was an increasing weakness, with a slight unproductive cough. Although his lesion changed, his complaint remained much the same; weakness. On admission his temperature was 100° going to normal in two days and remittent thereafter, varying between 99.6° and 102°, the average being 99.9°. The pulse rose to 120, the respirations to 34, averaging 26. After withdrawing 1,000 c.c. of sero-sanguineous fluid from the left pleural cavity on the seventeenth day, for an artificial pneumothorax, a thoracentesis was necessitated again on the thirty-fifth day with the withdrawal of 2,000 c.c. of bloody fluid, but without great relief. An increasing dyspnea and cyanosis supervened. His pulse became weak and more rapid, his right chest gave evidence of a beginning passive congestion, hemoptysis recurred with the loss of four ounces of bright red blood, and on the forty-ninth day of hospitalization, the patient expired. The laboratory reports gave normal urine; sputum negative for tubercle bacilli on fourteen occasions; blood Wassermann four plus; blood chemistry normal. The blood count and differential was: white blood cells, 10,800; red blood cells, 4,200,000; polymorphonuclears, 78 per cent. In the examination of the pleural fluid cancer cells were not observed. Guinea pig inoculation was negative for tuberculosis.

A post mortem examination was performed by Dr. William W. Hala, director of the pathological laboratories of the Kings Coun-

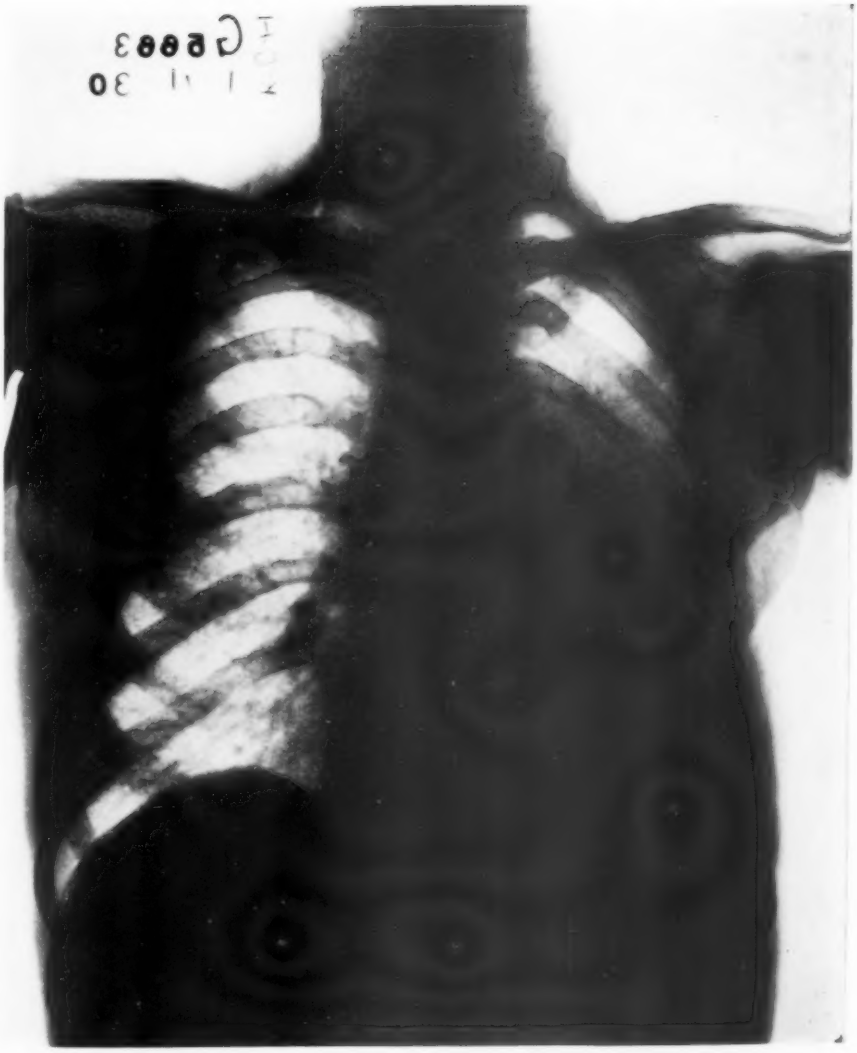

FIG. 1. (Nov. 13, 1930.) Absent aeration of the left lower lobe. Small effusion in the costo-phrenic space. Left chest smaller than the right. Moderate compensatory emphysema of the right lung. The left border of the heart shadow obscured by lung pathology. (Interpretation of the x-ray films was by Dr. Richard A. Rendich, roentgenologist at Kings County Hospital.)

FIG. 2. (Dec. 1, 1930.) Complete absence of illumination of the left lung field, consequent to a large collection of fluid in the pleural cavity; slight displacement of the heart and mediastinal contents to the right.

FIG. 3. (Dec. 1, 1930.) Oblique view after removal of one liter of sero-sanguineous fluid, air injection for study of pleural neoplasm—latter excluded by the normal pleural contour. Saccular aneurysm of the descending aorta now distinctly visualized. (From a P.A. view at the same time, the patient lying on his right side, the horizontal level of the remaining fluid was noted, with the absence of neoplastic mass of the parietal pleura.)

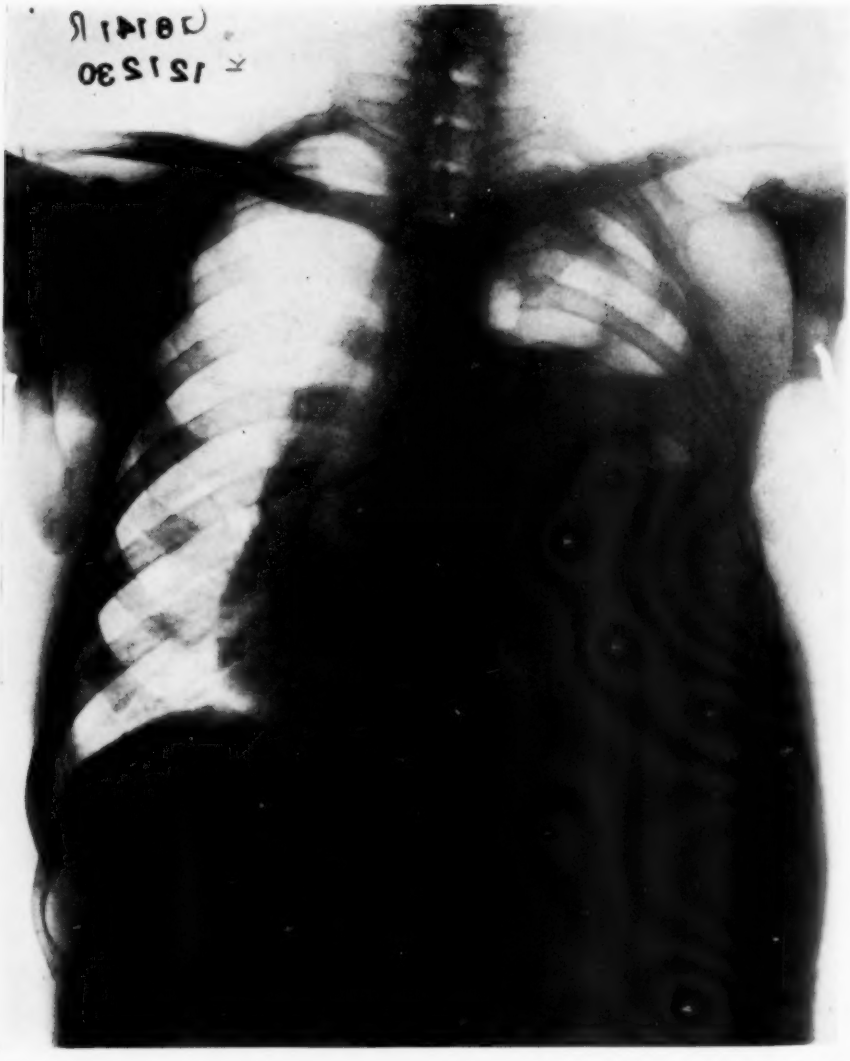

FIG. 4. (Dec. 12, 1930.) Further accumulation of fluid—several levels denoting small encysted collections.

ty Hospital, whose report follows, only data of positive interest being recorded here.

Main incision. The liver extended three inches below the costal margin in the midline. There were about 32 ounces of yellow fluid in the peritoneal cavity. The pericardial sac contained 25 c.c. of clear fluid. The left pleural cavity contained one quart of straw colored fluid .

The *left lung* was adherent to the chest wall, and from the base a copious thick purulent exudate escaped, coming from an area situated above the diaphragm and below the base of the lung, and encapsulated. The amount of pus present was about 400 c.c. On the posterior aspect of the lung and in the midline, in the region of the fifth and sixth dorsal vertebrae, there was noted a bulging of the descending branch of the aorta. Just below the bifurcation of the bronchi on the left side and postero-lateral, there was found a well circumscribed area the size of a golf ball, which contained about 10 c.c. of thick purulent material. In apposition to the upper portion of the lower left lung was found a sacculated aneurysm.

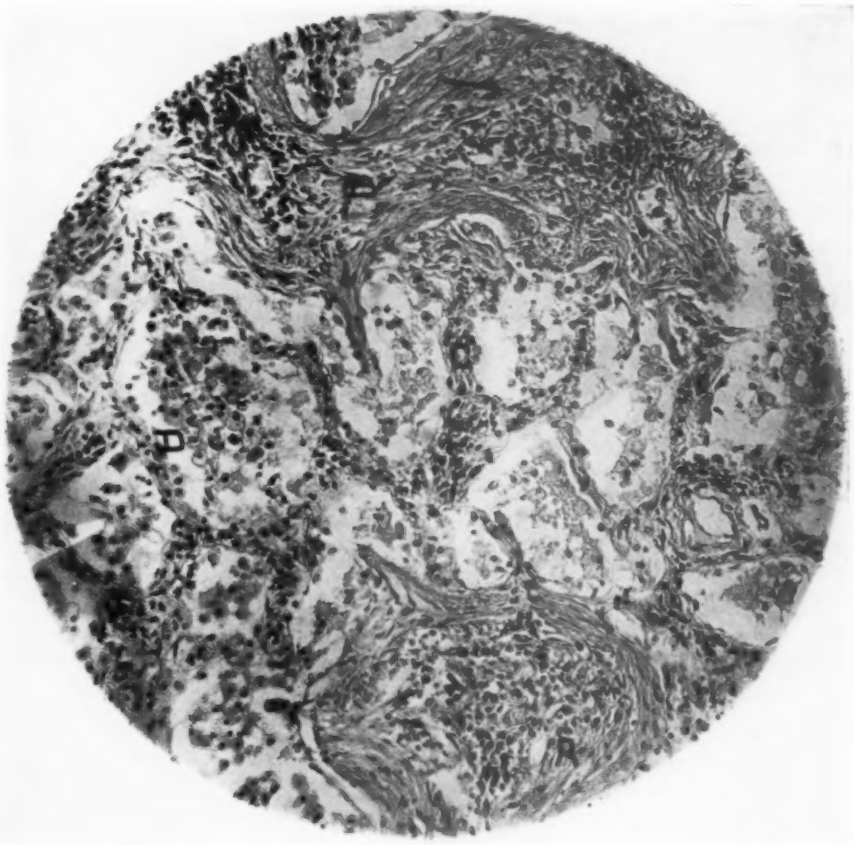

FIG. 5. Organizing pneumonia in syphilis. A. Alveolus entirely obliterated by fibroblastic tissue which is rather rich in cells of the small round and histiocytic type. B. Hyperplasia of interstitial tissue of the lung. C. A band of fibroblastic tissue bisecting an alveolus. D. An alveolus containing numerous large round cells, (exfoliated alveolar epithelia). The arrow points to an almost obliterated alveolus.

The *right lung* was heavy and of a dark gray color, the pleura was thickened and the lung substance had a "shotty" feel.

Lungs, trachea, esophogus, and aorta were removed in toto.

The trachea was dissected down to the bronchi and the left bronchus was dissected to its terminus. A seeping hemorrhagic tract was noted extending from a small pinpoint perforation of the aneurysm into the substance of the posterior portion of the lower left lung. The sacculated aneurysm, about the size of a lemon, was situated in the region of the fifth and sixth dorsal vertebrae.

The *aorta* had the described sacculated aneurysm, together with longitudinal striations of mother of pearl color and raised plaques with marked thinning of the wall.

The *lungs* on section showed diffuse tubercles and in the upper lobes of both lungs small cavitations and caseous material were observed. In the lower lobes of both lungs there was carnification. In the lower left lung there was an increased amount of stroma without aeration or crepitation. Near the base of the left lung, about one inch below the hilus, there was a grayish, rubber like mass the size of a five cent piece. The lungs presented the gray stage of hepatiza-

FIG. 6. Syphilis of the lung. The illustration shows productive inflammation induced by lues, practically no alveoli being preserved. At 'A' is an area of replacement fibrosis. The arrow points to a miliary gumma.

tion, and were firm throughout the whole middle and lower right lobes, and the upper left lobe.

The heart showed a myocardium of good quality, its valves and orifices grossly normal.

Microscopical Examination: The heart showed focal areas of interstitial myocarditis, and chronic epicarditis. The *lungs* showed a chronic fibroid productive pneumonia. In some areas this was still in the active stage showing marked organization. In other areas it was characterized by definite fibrillated replacement of the lung parenchyma, areas of bronchopneumonia and areas of abscess formation. Examination likewise disclosed the presence of numerous focal collections of small round cells, invariably along the pulmonary interstitial stroma. These collections were considered pulmonary gummata. There was no evidence of tuberculosis or neoplasm. In one section there was a rather large hemorrhage, occurring not only in the parenchyma of the lung proper, but in a rather large number of the bronchioles. It was highly probable the result of the ruptured aneurysm. The *spleen* showed a chronic splenitis, congestion and edema. The *kidneys* showed congestion, edema and an early vascular nephritis; the *aorta*, a syphilitic aortitis.

Anatomic Findings: Syphilitic aortitis; aneurysm of the descending thoracic aorta; chronic fibrinous pleurisy; empyema, encapsulated, left side; pneumonia, lobar type, gray stage; sero-fibrinous pleurisy of the right side with atelectasis of the lower right lung; atelectasis of the lower left lung; chronic passive congestion of the liver and spleen; acute nephrosis with vascular nephritis; hydropericardium; gumma of the left lung.

Syphilis of the lungs is rare and is seldom diagnosed clinically. In a search through the museums of the London Hospitals and the Royal College of Surgeons, Fowler[4] found but twelve specimens. Two of these cases were doubtful. At the Johns Hopkins Hospital, Osler[5] reported twelve cases out of 2,800 autopsies. Of these twelve, only four were acquired. Among 3,000 autopsies at the Massachusetts General Hospital, Lord[6] found only one case of acquired syphilis. Symmers,[7] in a study of 4,800 autopsy protocols, 314 of which showed lesions of syphilis, reported twelve cases and syphilitic pleural lesions in two more (Orsten[8]). Of 110,258 admissions to Kings County Hospital between and including the years 1919 to 1925, a period of seven years, the number of syphilitic patients, diagnosed as such by a positive Wassermann reaction, the history, or physical signs, was 5,695. Not one of these was diagnosed as syphilis of the lung. Dr. Henry Monroe Moses,[9] in a review of syphilis on his medical service at Kings County Hospital, between Dec. 1, 1923, and March 1, 1926, found in a series of 2,450 patients, 191 diagnosed as having syphilis. These patients were usually in the late stages of the disease, and sought hospital treatment because of some intercurrent ailment, or because of disability due to syphilitic involvement of some organ or organs of the body. Of the 191 patients in this series, the intercurrent illness was in the lungs in fifty. There were twenty-four with pneumonia, eighteen with lobar and six with bronchopneumonia. There were twenty-six with pulmonary tuberculosis. All of these patients were acutely ill on admission. There were seven deaths among these fifty patients, with forty-three who recovered or felt well enough to demand their own release. Of the seven deaths one was diagnosed as pulmonary syphilis, but unfortunately an autopsy was not obtained.

Syphilis of the lungs occurs as the congenital form—so called white pneu-

monia—which is a diffuse fibroblastic proliferation with an interstitial infiltration of small round cells, and is seen in stillborn babies or those dying shortly after birth. It may occur also in the acquired form as a chronic interstitial pneumonitis or syphilitic phthisis. It may occur as gummata, which is the usual type, and be rather sharply defined with radiating strands into the lung tissue. In the case reported here, we have a combination of these two forms. Rössle[2] differentiates four types of the disease. He distinguishes (1) the cavernous syphilitic phthisis; (2) the gummatous, coarse, knotty form; (3) the coarse lobulated syphilitic scarred lung (pulmo lobatus), and (4), the coarse syphilitic callosity without pronounced changes of the exterior form or shape.

According to Councilman,[10] the essential process in the production of a gumma in the lung is a pneumonia with fibrinous change in the alveolar walls, the whole subsequently undergoing caseation. The first step in the process is stated to be a hyaline degeneration of capillaries of the affected area. This is followed by atrophy of the alveolar walls. The alveoli become distended with large, pale, epithelial cells and fibrin; the cells also undergo hyaline degeneration, forming smooth bodies staining with eosin, and varying in size from one-half the diameter of a red blood corpuscle up to that of a large epithelial cell. The capillaries become converted into rigid tubes, and their lumina are much narrowed. Similar changes occur in the small veins and arteries. Immediately around the bronchi and arteries there is a formation of connective tissue, and here the alveolar walls show much thickening and contain many small round cells.

Gummata of the lung in elastic consistency resemble gummata found in other organs, occurring as nodules embedded in the tissues and surrounded on all sides by radiating fibrous strands. They may be single or in numbers, and may vary in size from that of a minute point as small as the smallest tubercle to the size of a hen's egg or larger, but the latter size is of rare occurrence. The central portion is firm, rubber-like, grayish white or yellowish like hard cheese. The necrotic, caseous part is analogous to that found in tubercles, but differing in its elastic, firm consistency and in its slighter tendency to liquify. A section through a gumma in the lung tissue might have exactly the appearance of one from a large caseous encapsulated tubercle in the same situation. Buhl[11] was the first to call attention to the persistence of smooth muscle fibers in the pulmonary callosity in chronic pneumonitis, while Tanaka[12] and Rössle[2] found not infrequently smooth muscle in the lung in syphilitic processes. Demonstration of the spirochete or of the tubercle bacillus would not settle the matter, for Schmorl[13] found spirochetes indistinguishable from *Spirochaeta pallida* in pulmonary gangrene and aspiration pneumonia, but these searches are notoriously discouraging of result.

The Wassermann reaction might afford important evidence. But as a rule the gross appearance and distribution of the lesions are found to be typical enough in each disease to allow one to

discriminate. Gummata have a strong tendency to heal, so that they are commonly found as disappearing centers of caseous material in great radiating scars, at times causing strictures, deformities, or obstruction, as in a bronchus. The presence of these lesions on roentgen ray examination, in the absence of obvious tuberculosis and when other signs of syphilis exist, warrants a diagnosis of gummata, but doubtless many of those described may have been localized encapsulated tubercles, or having in mind the apparent rarity of lung syphilis, gummata may have been diagnosed as tubercles.

"Gummatous infiltrations have a tendency to occur around the hilus of the lung and in the lower lobe. Occasionally areas of lobular hepatization are observed." (Hala[14]). The walls of the bronchi or the large vessels at the hilum of the lung may be greatly thickened by the process, the adventitia of the vessels suffering. By pressure of gummata or stricture of scars, obstruction of a bronchus may occur with atelectasis of the lung field, or the so-called indurative bronchiectatic type of pulmonary syphilis may supervene.

"Whether pneumonic or ulcerative forms of syphilis with cavity formation really exist is uncertain." (MacCallum[15]). Fowler[4] reports from the museum of Guy's Hospital a case of multiple gummata of the lungs, one of which was softening, breaking up and in the process of forming a cavity.

The confusion with tuberculosis makes this point difficult to settle (Flockemann[1]), especially since syphilitics are prone to tuberculosis (twenty-six of 191 on our service at Kings County Hospital during the years 1923 to 1925).

"In the diagnosis of pulmonary syphilis by the roentgen ray there is no characteristic picture."[16] This diagnosis should be made only by the history, a positive Wassermann reaction and the result of antisyphilitic treatment (Jaches[17]). "In the light of our present knowledge, even a tentative diagnosis of pulmonary syphilis is not warranted until every other possible type of pulmonary disease is excluded." (Allison.[3])

"The history of the case, the Wassermann reaction, the bacterial findings, the distribution of the lesions and the relation to lesions elsewhere, the size, consistence and gross appearance, the tendency to heal or break down, the continued absence of tubercle bacilli in the sputum, and least of all, the histologic structure, are the things upon which a diagnosis of syphilis in the tertiary stage may be based." (Allison.[3])

From the complicated pathological descriptions of pulmonary syphilis it is not difficult to realize that the clinical picture of this condition is not characteristic. The symptoms simulate those of pulmonary tuberculosis, and the diagnosis of tuberculosis is usually made. MacCallum's description of the tertiary stage of syphilis and how difficult it is to differentiate it from tuberculosis, especially in the lungs, may be a good explanation for syphilis of the lung being so rare as an autopsy finding (Orsten).

There is presented here a patient having syphilis of the lungs proven by autopsy findings.

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Observations on the Contour of Normal and Tuberculous Female Chests*†

By S. A. Weisman, M.D., F.A.C.P., *Minneapolis, Minn.*

In previous reports[1, 2] various diameters of normal and tuberculous male chests were compared. They showed that the tuberculous chest was more rounded, longer, and deeper than the normal chest, and that the vital capacity was about forty per cent greater in the healthy chest. In this paper the normal and tuberculous female chests will be compared. For normals, three hundred University of Minnesota girls were examined in the Spring of 1930 at the Women's Gymnasium. For tuberculous chests the data obtained on one hundred and thirty-three tuberculous women at Glen Lake Sanitorium, Oak Terrace, Minnesota was used.[2]

The ages of the normal girls ranged from sixteen to twenty-four years and of the tuberculous group from sixteen to sixty years. In order to make the comparison as close as possible, the tuberculous cases were divided into two groups; fifty cases representing the ages sixteen to twenty-four and eighty-three cases representing the ages twenty-five to sixty.

Thoracic Index

The average thoracic index, which is the ratio of the depth of the chest to the width, for three hundred University girls was 70.2. In comparing this average with those found by other investigators it was found that the Minnesota girls stand very high in chest development. Wilder and Pfeiffer[3] in reporting measurements on one hundred students at Smith College, stated that because the girls came from all parts of the Union it represented an average for the womanhood of the United States. The average thoracic index reported by them was 72.3. This represents a more rounded and less developed type of chest than that found for the University of Minnesota women by about three per cent. The same technique and statistical methods were used as described in my previous reports.

Again in 1929, also from Smith College, Steggerda, Crane and Steele[4] reported measurements on one hundred girls. The average thoracic index found by them was 75.2, which is 6.5 per cent deeper than that of our series. Their findings show a much deeper and more rounded form of chest.

The thoracic indexes for our series of tuberculous chests were:

Group I, ages 16 to 25, 73.3;

Group II, ages 25 to 60, 72.1.

After determining the probable error for the two groups and for the normals and computing the difference in terms of probable error, the findings

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are of some significance in comparing the two groups.

In comparing the normals with the first group of tuberculous chests, both ranging from sixteen to twenty-four, the difference in terms of probable error is 4.2. This indicates definitely that the tuberculous chest is more rounded than the normal.

ceptible to tuberculosis. Malone[5] and Hall[6] have shown by means of pantographic tracings that the better developed chest is flat and has a greater vital capacity. Müller[7] and Hutchinson[8] have shown that the chest of the fetus is deeper than it is wide. Scammon and Rucker[9] have demonstrated that a baby's chest at birth is nearly round,

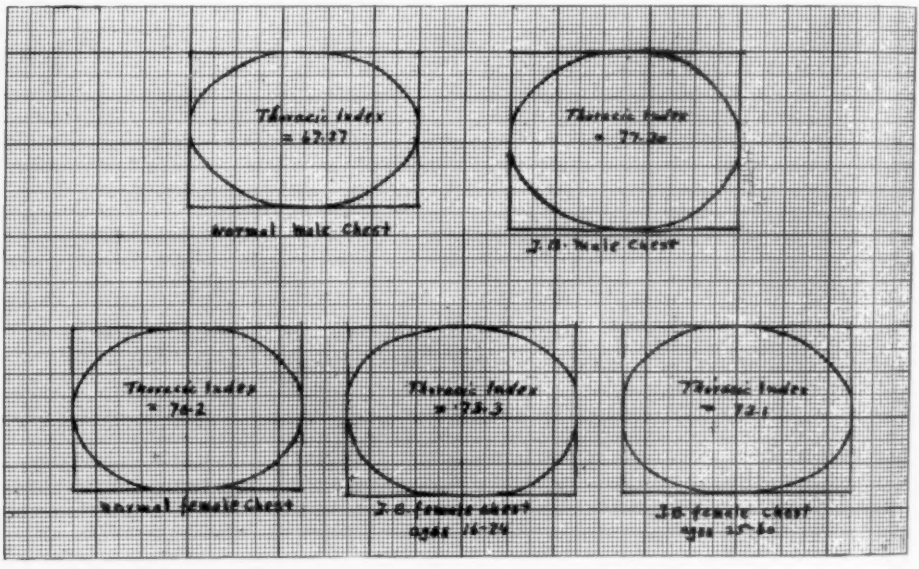

FIG. 1. Diagrammatic sketches of chest diameters.

In comparing group II, ranging in ages from twenty-five to sixty, with the normals, the difference in terms of probable error is probably, but not certainly, significant, namely 2.67.

The above figures tend to show that the older tuberculous group have a flatter and better developed chest than the younger group. This fact emphasizes a point brought out in previous reports,[1, 2] that the deeper, rounded chest is an undeveloped one; it is the primitive chest; it has a lower vital capacity, and it is, perhaps, more susthat it gets deeper after the first respiration and that it is about three months or more before it reaches the thoracic index it had at birth. Zeltner[10] has shown that the chest becomes more flattened up to the twentieth year, and Stewart[11] states that the vital capacity at about the twentieth year is the greatest. At this age the chest begins to change toward the infantile type and slowly assumes a more rounded form until development ceases at about the sixtieth year, as shown by Weisenberg.[12]

Therefore one should expect an older person to have a more rounded chest than a younger one, and an older individual with tuberculosis should surely have a more nearly round or deeper chest than a younger one afflicted with the same disease. Our findings, however, show the opposite to be true. The older tuberculous group have flatter and better developed chests than the younger tuberculous group. This perhaps indicates that the nearer round or the less developed the chest is in early life, the more liable is that individual to develop pulmonary tuberculosis. The flatter chest, like that found in group II, probably means that that chest was more resistant to tuberculosis; it had a better vital capacity and warded off the disease until later in life.

Draper[13] in his book, "Human Constitution," gives the measurements of twenty-eight tuberculous female chests. The thoracic index was calculated as 74.5 which is a more rounded chest than either of our groups. The difference, as with other investigators mentioned above, may have been due to the technique used. In our series the normal female chest was found to be more rounded than the male. Rodes[14] showed that the female negro chest is narrower than the male. Draper, too, showed that the female white chest is narrower than that of the male. Jack-

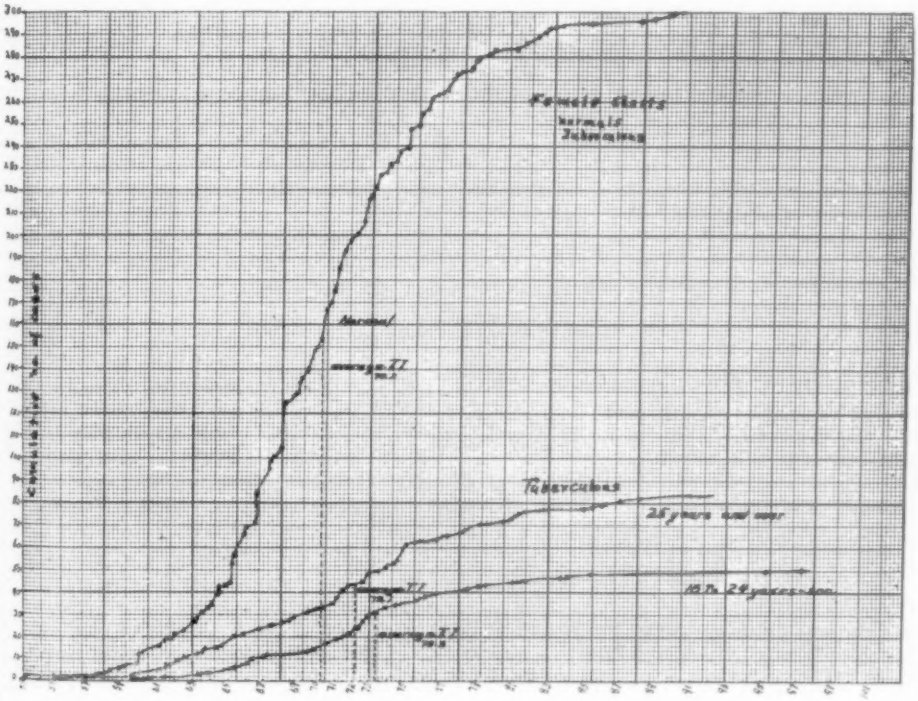

FIG. 2. Cumulative number graphs of thoracic indexes in groups of women considered.

son[15] has shown that the average chest expansion and vital capacity in the female is less than it is in the male. In our series[2] of six hundred and five University of Minnesota male students the thoracic index was found to be 67.27.

The Subcostal Angle

The subcostal angle in the normals and two tuberculous groups was measured. A definite difference between the normal and the diseased chests was found. The average for the three hundred normal girls was 75.9° and for group I of the tuberculous, 68.7°. The difference in terms of probable error is 5.46. The average for group II of the tuberculous is 71.1°. The difference in terms of probable error is 4.8. These findings, too, are in accord with those of the thoracic index; that the older tuberculous group has a better developed chest than the younger group.

Age

The average age for the three hundred normal girls was 18.7 years, and for the tuberculous group I, 21.1 years. The range in both of these series was between ages sixteen and twenty-four. Tuberculous group II ranged from twenty-five to sixty years. The average age was 31.7 years.

Comment

As was emphasized in the previous reports, perhaps it is wise to again stress the importance of measuring, as a routine, the chests of children. In this way one is more able to find those with undeveloped chests; chests that are perhaps more susceptible to tuberculosis. Proper exercises can help improve the undeveloped chest. Götz[16] working with children, and Turner's[17] recent report of results on young female adults show that proper exercises increase the vital capacity of the lungs.

Conclusions

1. Female tuberculous chests are shown to be more nearly round and deeper than the normal.
2. There is a possibility that the rounder the chests the earlier in life is one apt to contract tuberculosis.
3. There is evidence that proper exercises in early life can stimulate chest development.

Table I

Summary of Indexes of Thoracic Measurements (Female)

Type	Thoracic Index	Subcostal Angle	Average Deviation		Probable Error		Difference in Terms of Probable Error		Age	
			Thoracic Index	Subcostal Angle	Thoracic Index	Subcostal Angle	Thoracic Index	Subcostal Angle	Range	Average
Normals	70.2	75.9°	4.46	4.48	±0.229	±0.256			16 to 24	18.7
Tuberculous*										
Group I	73.3	68.7°	3.00	10.2	±0.695	±1.33	4.2	5.46	16 to 24	21.1
Group II	72.1	71.1°	6.00	9.81	±0.675	±0.978	2.67	4.80	25 to 60	31.7

*The thoracic index of the tuberculous female chests reported in my previous paper (2) is 72.6 instead of 72.1. (Error in printing.)

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American Mountain Tick-Fever—Semiography and Nosology

With Remarks on Pathology and Treatment*

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THE tick borne fever of the mountainous areas of western North America has on several occasions since 1850 been the subject of brief remarks and of other publications of a more comprehensive character. It cannot be said, however, that the disease has ever been adequately described. All of the early and more recent descriptions of the disease show the want of knowledge concerning its etiology, its epidemiology, and its immunological relationships. Indeed, the published observations on the American mountain tick-fever have been for the most part either fragmentary or confused by the observers' preconceived ideas.[3] Nevertheless, it would be unjust to carp at the memoirs on mountain fever that were written by W. T. Ewing,[4] by Roberts Bartholow,[5] by John J. Milhau,[6] by Charles Smart,[14] and by Charles F. Kieffer,[40] as those observers deserve great credit for recording accurate and succinct descriptions of the disease as observed by the unaided eye.

What has been most remarkable is not the essential inadequacy of early descriptions of the mountain or non-exanthematic tick-fever, but the fact that it has been necessary for the present writer to re-discover it, at least from the aspects of calling attention to its existence and of establishing it as a separate disease entity[47,48].

The non-exanthematic tick-fever of the mountains has a characteristic clinical course, but a course that varies with its virulency to range from a mild, remittent-recurring type of fever to a severe typhoidal form. In the latter type the fever is so continued as to virtually obscure the remittent and recurrent characteristics. Some cases commence with marked remittency of the fever but pass into a continued fever, high or low, with more or less stuporousness and prostration. These two types, the recurrent and the continued (with more or less remissions) have been noted by most observers. They were treated as separate types by Kieffer,[40] and will provisionally receive like treatment by us, but with the qualifying remark that the continued form is very probably tularemia, but has not yet been proven to be so. These types are not absolutely fixed but are observed to somewhat intergrade with one another, which lends color to the belief that mountain fever may occasionally take a continued fever

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form. In some localities the recurrent type of the fever is more characteristic than in other localities, thus in the Black Hills and in Wyoming the fever is predominently recurrent whereas in Colorado and Utah it is more commonly observed to have one well marked major episode followed by one, or at the most two, very minor exacerbations. A distinct tendency to a remittent and recurrent character can, however, be almost always made out even in the so-called continued type, and similarly in the Colorado-Utah type with one major episode, the recurrences are often so minor in character as to frequently attract no special attention.

Clinical Course

With but very few exceptions, the *incubation period* is four days. Longer incubation periods have been observed but seven days is the longest authentic instance. Kieffer observed a case with an incubation period of three days.

The *prodromes* consist of extreme weariness and faintness (languor), loss of appetite, muscular weakness, and an "empty or sinking feeling" in the epigastrium. They merge with and continue throughout the onset, which usually occurs within two to eight hours after the first appearance of malaise.

The *onset* is sudden with profound chilliness (but with no rigor) lasting from one to two hours and alternating with occasional flashes of heat. The cold stage is suddenly succeeded by a rapid accession of fever, accompanied by excruciating pains in the back and loins, and to a somewhat less extent throughout the body, especially the posterior cervical region and the extremities, with occasional cramping of the legs. A diffuse dull headache, chiefly frontal, occurs commonly but is rarely severe, although occasionally intolerable. The muscles become tense and the small muscles twitch, particularly the orbiculars and those of the face. Nausea is usually noticed. Vomiting is usually absent in adults but may occur in children, although not as commonly as in other fevers. The face becomes flushed, the facies tense and troubled, the skin hot and dry, the tongue red and swollen ("strawberry tongue"), the conjunctivae very markedly congested, but lachrymation does not occur.

The pulse, at first rapid (85 to 90), full and bounding, becomes, after a few hours, very rapid (120 to 130), thin and tense and continues so for twenty-four to forty-eight hours.

The *fastigium* is characterized by a fever of 103.4° to 104.6° F., by the patient lying motionless, for fear of pain on motion, but with a clear though restless sensorium, sleep being impossible, or broken and troubled. Breathing is shallow, a trifle quickened and sighing. Meteorism does not occur.

Constipation is the all but invariable rule throughout the course of the disease, the patient usually going four or five days without defecating. Should evacuation take place spontaneously there is great tenesmus and pain in the anus, the feces being scybalus and covered with mucus, frequently blood tinged.

The tongue, at first congested ("strawberry tongue"), becomes swollen, large and flabby, with a

very thick, moist bluish-white glaze of pasty (cheesy) consistency to the very tip, latterly cracking down the center. The tongue is tremulous when protruded.

Urination is impeded by muscular tenesmus, or almost entirely suppressed; or if any urine is passed it is scanty and highly colored, causing scalding when voided.

The liver and spleen, and all the muscles are tender to touch, the deep bone and joint and muscle pains continuing, but without redness or swelling of the joints.

Perspiration begins gradually and continues scanty for twenty-four to thirty-six hours, to end in a moderate (but seldom drenching) sweat coincident with the rapid lysis that ends the febrile period at the end of the second or early part of the third day. With defervescence, the congestion of the conjunctivae and face, the headache, and muscle aches, subside, the patient being left free from pain, but prostrated.

No erythema or other cutaneous manifestation develops at any time, but after the final defervescence a fine branny desquamation of the skin may occur.[4] The latter is not common and seems due to medication with antimony or arsenic.

On the fourth day after the first accession of the fever, the patient feels very well, and desires to be up and about; but on the fifth to sixth day in a large proportion of the cases the fever, weariness, and muscle pains return, but with much less severity than at first, and last only from twelve to eighteen hours. After this recurrence subsides the patient convalesces slowly, except that there may be one or two more recurrences of the fever to a slight degree.

On and after the fifth day the countenance assumes a dull despondent expression; the face a dirty yellowish hue, with a tinge of dark brown in the cheeks.

Usually the total duration of the disease is eight or nine days, although not uncommonly ten days. Very exceptionally it may be as much as fourteen to twenty-six days; especially so when of the continued fever type. Some slight cases run their course in five or six days, but some of the latter cases, if observed under the advantages of hospital care would be recognized as of the eight day duration.

Convalescence is characterized by marked anemia, by a persistence in the sensation of weariness throughout the whole body, and weakness and stiffness of the joints of the extremities. The carpo-metacarpal, metacarpal-phalangeal, and phalangeal joints are sometimes especially stiff and painful.[14] Bartholow believed that "In many instances, the first evidence of commencing convalescence, was a violent pain in the soles of the feet, increased at night."[5]

Variations in the Clinical Course

The prodromes very exceptionally drag on for two or three days (in cases with a seven day incubation period), but more commonly than being prolonged they are less than three hours in duration, the disease striking suddenly. Very frequently slight muscle pains are noticed during the prodromal stage, but they do not become excruci-

ating until the onset. The onset is never free from pain, but in mild cases the muscle and bone pains develop gradually after the initial chilliness has passed off. The initial epigastric distress is sometimes described as a hunger pain.

The chilliness is sometimes so slight as to escape the observation of the patient. Sometimes the initial chilliness is distinctly intermittent, alternating every fifteen minutes to one hour with like periods of sweating and a sense of heat; the intermissions occurring approximately hourly for the first twenty-four hours, or until the fastigium is well established.

Nausea is very variable, in some outbreaks being a prominent symptom and occurring frequently before or with the onset of the chilly (initial) stage. If it has not occurred during the invasion it is extremely unlikely to occur during the fastigium. Vomiting occurs fairly frequently in children, but not as commonly as in other fevers. In adults, vomiting almost never occurs, or at least not without great difficulty (about ten per cent of the cases) due apparently to a cardiospasm. On the other hand, there are exceptional cases in adults in which vomiting occurs repeatedly with each onset of a febrile paroxysm.

The headache is occasionally described by patients as being intolerable, and feeling very much as if the head were being mashed between rollers.

The perspiration is occasionally established early and gives a cold clammy feel to the skin after the initial chilliness has existed for an hour or so. Equally frequently the skin is hot and dry, and moist and cool by turns, alternating at eight to twelve hour intervals. The perspiration of the rapid lysis is always marked, and occasionally deserves the characterization of a drenching sweat, but usually it does not. On the whole, very noticeable hyperidrosis is not a characteristic of mountain fever.

A low, more frequently violent, delirium may occur during the fastigium, and recurrently at the height of each febrile paroxysm. It is most likely to be observed in alcoholics, nephritics and others with an impaired metabolism.

Occasionally, but very exceptionally, the bowels become spontaneously relaxed after the second or third day (once only as a rule). The stools, dark brown and watery and containing scybalous masses, are extremely offensive in odor. Meteorism has been observed in exceptional instances.

In mild cases the patients do not go to bed but drag around complaining of feeling "out of sorts", stupid and sleepy, with loss of appetite, constipation, and aches and pains in the bones and joints, or "neuralgias".

The fever typically lasts for forty-eight hours and recurs with less intensity after an intermission of forty-eight hours, the paroxysms of fever and intermissions being each of two days duration, but the fever becomes progressively less severe with each remission. A general type of regularity can be made out, and some of the early epidemics described by army surgeons evidently ran with few exceptions to the regular type encountered during the epidemic described, but on the whole mountain fever is not as regularly remittent and recurrent as malaria is intermittent. Irregularity of the

remittences and recurrences is common so that mountain fever is best described as a fever distinctly irregular but with marked tendencies to a regular type.

The various observed febrile courses can be reduced to a graphical portrayal as follows:

Numbers in parentheses represent days of fever; other numbers, days of intermission. Accents represent degree of intensity: two, severe; one, moderately severe; none, mild. Figures: (2) (1) and (1) (1) mean that a nearly complete or very decided remittence occurs between the days indicated but lasts for only a relatively brief time.

v=vomiting; d=delirium; ....=convalescence; x=one, two or three additional (usually mild to moderately severe* recurrences).

(2)″ 2 (2)′ 2 (1) .... 16 per cent
(2)″ 2 (1) 3 (1) .... 5 per cent
(2)″ 2 (2)′ 2 (2) x .... 6 per cent
(2)′ 2 (2)″ 2 (2) x .... 3 per cent
(2)′ 2 (2)″ 2 (1) .... 5 per cent
(2)′ 2 (2)″ 2 (1) 1 (1) .... 3 per cent
(2)″ 2 (1)′(1) 2 (1) 1 (1) .... 10 per cent
(2)″ 2 (1)′(1)′ 2 x .... 5 per cent
(1)″(1)′ 2 (1)(1) 2 (1) .... 5 per cent
(1)″(1)″ 2 (1)′(1) 2 (1) .... 5 per cent
(1)″(1)″ 2 (1)′(1) 2 x .... 2 per cent
(3)″ 2 (2)′ 1 (1) .... 10 per cent
(2)″(1)′ 2 (2) 1 (1) .... 6 per cent
(2)″(1)′ 2 (2) 1 x .... 2 per cent
(2)″(1)′ 2 (1)(1) 1 x .... 2 per cent
v(2)″ 2 (2)′ 2 (1) .... 2 per cent
v(3)″ 2 (2)′ 1 (1) .... 1 per cent
v(2)″(1)′ 2 (2) x .... 2 per cent
v(2)″ 2 v(1)′ 2 (1) .... 1 per cent
(2)″d 2 (2)′d 2 x .... 3 per cent
(2)″d(1)′d 2 (2)′d 2 x .... 1 per cent
(2)″d(1)′d 2 (1)d(1) 2 (1) .... 2 per cent
v(2)″d 2 (2)′d 2 x .... 1 per cent
v(2)″d(1)′d 2 (2)′d 2 x .... 1 per cent
v(2)″d(1)′d 2 (1)d(1) 2 (1) .... 1 per cent

*These percentages merely represent our impression as to the approximate frequency with which the different combinations of remittence and recurrence occur. It is not to be supposed that the above groups exhaust all combinations that may be encountered, but they well exemplify the "irregular regularity" of mountain fever.

The 'days' as given, particularly as referring to the days of intermission, are not to be understood to be exact periods of twenty-four hours. In fact there is not as exact a diurnal cycle seen in mountain fever as in the paludisms.

Physical Findings

The marked conjunctival engorgement has already been noticed. Epistaxis may occur but is decidedly uncommon. The fauces are normal or slightly injected, but never extremely hyperemic. There is no buccal enanthem. Cervical and other external adenopathy is not noticeable.

Chest findings are not a part of the disease but a secondary bronchitis sometimes follows the disease when inclemencies of the weather have been experienced during the pyrexial period.

Abdominal discomfort, always present to some extent, is vague, variable, and inconclusive. Rigidity and hyperesthesia, and pain on deep palpation, are to be discounted as the patient is hyperesthetic and tense over the whole body. The spleen becomes barely palpable early in the course of the disease but does not become more than slightly enlarged. The liver is always tender and sometimes a trifle enlarged. A distended bladder may be encountered.

Diagnosis

Mountain fever is particularly devoid of characteristic (pathognomonic)

physical findings. Not one of the physical findings may not be found in some other fever or even group of fevers. The nearest approach to an exception is the tongue, which is fairly characteristic, and yet by no means pathognomonic. Hence diagnosis must rest, in the present state of our knowledge, upon a consideration of the attending circumstances (locality, season, history of tick bite or of exposure), exclusion of other possibilities, and upon the appearance of the clinical phenomena as a whole; the latter being unique when considered with respect to their syndrome and evolution. The differentiation from the two most clinically similar diseases, dengue fever and relapsing fever, will be discussed under the heading of nosography.

Owing to the history of tick-bite, the disease that gives rise to the greatest practical difficulty is spotted fever of the Rocky Mountains. Where mountain fever and spotted fever co-exist locally it is not to be supposed that mountain fever can always be distinguished from spotted fever during the first two days of its course. After the second or third day, however, spotted fever should be excluded by a consideration of the following clinical differences:

Spotted fever has a somewhat less stormy onset with a more protracted accession of fever, with a continued and prolonged (and usually ultimately higher) character, with only slight or moderate remissions. Important, also, are the lesser intensity of spotted fever's muscle and bone pains, the stupor developing in spotted fever after the second or third day (even in mild cases, although not in the mildest, ambulatory, type). The lesser degree of nausea but the more marked cyanosis and hyperidrosis in spotted fever are usually noticeable after about thirty-six to forty-two hours. The puffy facies of spotted fever contrasts with the pinched facies of mountain fever. The spotted fever case lies relaxed, "log-like"; the mountain fever case is more tense, for fear of pain on movement. In spotted fever insomnia is all but absolute, in mountain fever it is only relative, sleep occurring fitfully. A dry cough, with or without a small amount of very tenacious mucus, occurs with the prodromal stage or onset of spotted fever but is not present at all, or only very late, in the course of mountain fever. By the fifth or sixth day the spleen is larger in spotted fever than in mountain fever. The tongue tends to remain more swollen and much less coated in spotted fever than in mountain fever, and frequently cannot be protruded in spotted fever. Finally one of the most helpful and invariable differences is the absence of the characteristic lenticulo-macular (initially slightly raised) erythematocyanotic (frequently hemorrhagic) exanthem of spotted fever, which first appears on the ankles and wrists and extends upwards to cover the trunk but (usually) spares the face. A perhaps inconstant difference, and yet one helpful at times, in the early course of the disease, is that the abdominal symptoms of spotted fever at onset suggest an acute cholecystitis (or hepatitis) whereas those of mountain fever suggest a cardiospasm with ulcer or gastric dilatation symptoms.

As the continued form of mountain fever may be very difficult or impossible to distinguish from mild or aberrant types of typhoid fever and the paratyphoids, no attempt will be made to describe the (inconstant) clinical differences except to call attention to the semi-liquid stools, the occipital instead of frontal headache, the small, dry tongue, the ultimate bradycardia, the greater wasting of body tissues, the scaphoid abdomen, and the odor of body and breath encountered in the enteroidea group. It is of course understood that agglutination tests or

blood cultures may be necessary for definitive diagnosis for both the enteroid group and tularemia, which will also frequently have to be taken into consideration.

Acute epidemic influenza has undoubtedly been mistaken for mountain fever, and accounts for some of the so-called mountain fever reported in the winter months.[10] The much greater irregularity of its remittances, the profuse lachrymation, rhinorrhea and sneezing, the acutely congested pharynx, with frequent secondary otitis and nasal sinusitis, the symptoms of bronchial involvement, the larger, more compressible pulse, the more superficial character of the muscle aches, with particular predilection for the intercostal and cervical groups, the (occasional) singultus, and the tendency to a critical or epicritical diarrhea, make up a clinical picture that ought not to be mistaken for mountain fever.

Regional septic processes, perinephric abscess for example, may cause a heavily coated tongue and an irregularly-regular type of remittent fever, but with them the generalized intolerable "break-bone" pains are absent, whereas a sharp, knifestab-like, or deep "hot iron," regional pain is present.

Pathology

No necropsy material has been available.

The *urine* shows nothing noteworthy. During the prodromal stage the patient often passes much clear urine of low specific gravity. The urine drawn during or after the fastigium is of high specific gravity, acid, and with excess of pigments and bladder epithelium, but is free of sugar, acetone, bile and albumin unless some renal impairment has already been present. Hyaline casts may be found.

The *blood* shows a profound change, especially so considering the intensity and short duration of the fever. The red cells become greatly reduced; in severe cases to 3,000,000 or even to 2,500,000, which is less than fifty per cent of the normal red count at high altitudes. Hemoglobin diminishes in ratio with the red count, the color index remaining 1, although when regeneration commences the red cells increase at greater rate than the hemoglobin, the index falling below 1. The anemia is predominantly a toxic, aplastic, rather than a hemolytic anemia, although the muddy color of the face late in the disease suggests the occurrence of some hemolysis.

Neither a leucocytosis nor a leucopenia develops although the white count tends to range a trifle above normal. The differential count shows a constant increase of the large mononuclears (15.0 to 16.5 per cent) with lymphocytes around ten per cent and eosinophiles under 1 per cent. The large mononuclears are increased absolutely as well as relatively, a condition also noted in spotted fever.

The absence of jaundice speaks for very little derangement of the liver or bile ducts.

Numerous cases of mountain fever studied repeatedly in the laboratory at the Post Hospital of Fort D. A. Russell have shown conclusively that the serum of mountain fever does not contain the typhoid organism nor agglutinate the latter.

So far, numerous efforts to establish the disease in guinea-pigs have failed.

This is one of the best evidences that the virus is distinct from that of spotted fever.

Prognosis

In mild cases convalescence is more rapid than in dengue but in severe cases it may be prolonged for weeks. Organic complications or local sequelar infections do not develop, except very rarely the cutaneous pyodermata in those who are uncleanly and already debilitated, with chronic foci of staphylococcic or streptococcic infection.

Mortality is not indisputable, but is extremely rare at best. In the early literature several deaths attributed to the continued form of the disease were recorded but the true nature of these cases is uncertain, much of the severe, so-called mountain fever undoubtedly having been typhoid fever[16, 17, 20, 26, 33] or tularemia. And yet there is no reason why a severe case of mountain fever should not carry off a debilitated individual or a patient embarrassed by cardiac insufficiency.

Treatment

The patient should be put to bed and made comfortable, blankets and hot water bottles, or tepid sponging, being used according to the stage of the disease. A large hot toddy or hot lemonade is grateful. Emetics were formerly much employed but are of doubtful value unless retching is continued and troublesome.

During the initial episode, and severe recurrences, morphine should be given hypodermatically in effective dosage every four hours, as it alone relieves the excruciating bone and muscle pains. For the milder recurrences, acetphenetidin or the less efficacious acetylsalicylic acid will be satisfactory; acetanilid should be avoided. To quiet the sensorium a hypnotic such as barbital, fifteen or twenty grains, should be given and repeated as necessary, or a bromide in sixty grain doses is usually sufficient; chloral hydrate should be avoided. A collyrium of half saturated boric acid solution to which a fifth-part of epinephrin chloride solution (1-1000) has been added is grateful to the eyes. If photophobia is noticeable a dark cloth should be placed over the eyes unless the room can be completely darkened. An alkaline, antiseptic mouth wash should be prescribed for frequent use. An isotonic solution made of equal parts of salt and baking soda does equally as well. The bowels should be moved, preferably by enema in the early stage of the disease, but, as soon as the onset has passed off, by a saline cathartic or by a hydragogue cathartic pill, such as the calomel-rhubarb-colocynth pill, repeated daily for two or three days. The bladder should be watched, and if necessary to prevent distension, the urine should be drawn. Digitalis is rarely indicated, as in spotted fever, except in the continued fever type of the disease, when it is well to start it early.

Other than to place the patient on a sensible fever diet, with thought being given to the constipation, no special dietary restriction is necessary. During convalescence, the severe anemia should be combated by means of an iron, arsenic and copper hematinic, and cod liver oil according to indications. For ending the persistent joint aches, a short course of sodium or potassium iodide in large dosage, with or without

a little Fowler's solution, is distinctly valuable.

There is no known specific for the disease. Quinine was formerly much employed, and much abused, but impartial observers as Milhau[6] and Kieffer,[40] early discovered that it not only had no effect on the fever but in large doses did harm by increasing the subjective symptoms.

Kieffer attached considerable value to subcutaneous injections twice daily of one or two cubic centimeters of a one per cent solution of sodium arsenite to which four per cent cocaine hydrochloride was added to control the pain. We feel that neither the sodium arsenite nor sodium cacodylate injections are necessary, but that Fowler's solution by mouth in appropriate but fair-sized dosage will do as much good. It should not constitute routine treatment but should be reserved for cases that continue to run a fever after the seventh day, or have protracted muscle aches and joint pains.

Nosology

The non-exanthematic tick-fever of the mountainous West evinces a syndrome not unlike, in some respects, the clinical course of two diseases, each of which is the type of a different disease category, namely dengue fever and relapsing fever. As to which category it is more nearly correct to assign the American mountain fever can be determined for clinical purposes, but for a final assignment on the basis of bacteriological and pathological evidence it will be necessary to await the study of the virus of mountain fever. Other fevers have a recurrent clinical course, such as undulant fever (brucelliasis, melitococcemia) but it would for the present seem to labor a point to go beyond a consideration of mountain fever's two most presumable cognate affinities.

Compared with the relapsing fever group we find that mountain fever and the relapsing fevers are arthropod-borne diseases; that neither are invariably exanthematous; that both are characterized by a marked secondary anemia, and that both have well marked recurring or relapsing characteristics. There are these differences however: the relapsing fevers are caused by *Spirochaeta* that are not difficult to recover from the blood at some time during the clinical course; they are notable for being truly relapsing virtually to their final episode, which is seldom the second or third relapse. They prostrate, but do not cause the profound bone, joint and muscle pains that are practically a fixed character of mountain fever. The relapsing fevers have a *comparatively* insidious onset and a much greater interval between relapses than does mountain fever. They frequently cause a bronchitis and a disorder of the cornea not observed in mountain fever. Finally they are of the bilious type with icterus occurring in one-fourth to one-half of the cases, according to the severity of the epidemic.

Compared with dengue fever we find that mountain fever is of like short duration; that it is, like dengue, more properly called recurrent than relapsing, and that the disease is of equally, or nearly equally, abrupt onset. It has in common the "break bone" symptoms, the congestion of the conjunctivae, and the absence, or virtual ab-

sence, of bronchial involvement. Mountain fever differs from dengue fever in lack of a palmar and plantar erythema and a generalized erythematous or polymorphous eruption. Also there is not the cervical adenopathy that one finds in thirty per cent to seventy per cent of the cases of dengue. Lachrymation occurs in dengue but not in mountain fever. The tongue is not greatly different in the two diseases although more heavily coated in mountain fever. Constipation is not so absolute in dengue, purging being observed in about one-third of the cases. Mountain fever differs in producing a far greater anemia and in not being accompanied by the characteristic and well marked leucopenia of dengue. In both fevers there is an increase of the large mononuclears, but the eosinophiles are increased only in dengue.

From the above comparisons it will be observed that mountain fever has an intermediate position between the short fevers of the dengue type and the protracted fevers of the relapsing fever group. Similar in some reactions to the one and yet in other reactions more closely resembling the other, it serves almost as a 'connecting link', were one to take cognizance of the theory that dengue is a spirochetal disease. We do not wish it understood that we are asserting a theory of mountain fever's spirochetal nature, but when one considers the comparative difficulty with which the *Leptospira icterohemorrhagica* is recovered from the blood in the spirochetal type of infectious jaundice, a protozoan of some such character may occur in the blood of mountain fever, and yet have escaped detection. In this connection it should not be forgotten, however, that Noguchi recovered from the mountain fever tick, *Dermacentor andersoni,* a filterable virus that was demonstrated not to be the virus of spotted fever.[44] Whether the filterable virus recovered by Noguchi is that of mountain fever remains to be ascertained.

Apparently mountain fever has closer affinities to dengue fever than to relapsing fever but whether the resemblance is sufficient to warrant grouping it provisionally with dengue fever may well be a matter of opinion; but, keeping in mind that it is but tentative, it would seem permissible clinically to group mountain fever with dengue fever.

The Continued Form of Mountain Fever—Possibly Tularemia

Appearing from the outstart as a continued fever, or more commonly becoming a low continued fever after an initial phase with remittences, this type of mountain fever tends to a longer course than the remittent type. Two weeks to twenty-six days is the usual duration, but in Colorado and Utah, and occasionally in Wyoming, a shorter fever of nine or ten days may be predominantly of the continued type, although some minor degree of remittence can usually be made out.

The continued form is somewhat typhoidal as far as the fever and the nervous system are concerned, but with those exceptions there is not a close resemblance to typhoid fever. The continued form seems to be a lower grade of infection limited largely to the blood stream. In it the muscle-bone-joint pains and other localizing phenomena are not nearly so severe. Towards the

end of the disease the patient is weaker and more emaciated than after the short recurrent type, but convalescence is not thereby prolonged, it being by no means unusual for these cases to convalesce somewhat more rapidly than those who had a typical recurrent type of American mountain tick-fever but continued to have persistent bone and joint pains during convalescence.

There is very considerable reason to suspect that the so-called continued type may not be due to the virus of American mountain tick-fever but to a *Bacterium tularense* infection. The local lesions, acute but indolent skin ulcers at site of tick bites, typical of tularemia, occur following tick-bite in some of these cases, but they certainly have not been observed in all.

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Bilateral Double Kidney With Duplication of Ureters*

By Robert M. Lintz, M.D., *Brooklyn, N. Y.*

Anomalies of the various parts of the body are not infrequent, but with the exception of those of the spine those occurring in the genito-urinary tract are most often noted.[1] One of the rather frequent anomalies of the genito-urinary tract is that of double kidney with duplication of renal pelves and of the ureters. Young and Davis[2] state that the *condition* of double kidney and ureter is not so rare, but that the *recognition* of the condition is extremely rare. They believe that anomalies of the kidney and ureter are much more frequent than is generally appreciated and that among such kidneys a relatively large number show pathological changes, malformation predisposing to disease.

Various types of duplication of the ureter with the accompanying double kidney have been reported from time to time. It is possible to have unilateral involvement with the other side normal or both sides may be changed from the normal. In the condition known as complete duplication of the ureters there are separate bladder orifices for each ureter with the ureters running from the pelves to the bladder. This means that where there is complete bilateral duplication there are four ureteral orifices present in the bladder. In the incomplete form of duplication there is union of the duplicated ureters somewhere between the emergence from the pelves and the entrance into the bladder. In these cases the bladder may present the usual appearance as far as the ureteral orifices are concerned. Where there is complete duplication of one side and either a normal opposite side or incomplete duplication of the opposite side three ureteral openings into the bladder will be present.

Thompson[3] reported fifteen cases of duplication of renal pelves and ureters in 1735 consecutive autopsies at London Hospital and three cases in 11,133 consecutive autopsies performed at Guy's Hospital and at Victoria. It is interesting to note that of the eighteen cases of duplication sixteen were in females. Mauclaire and Séjournet[4] found the ratio of occurrence in females to males to be 7 to 3. Thompson's observation is at considerable variance from those of some of the earlier investigators. Wagner,[5] Poirier,[6] and Bostroem[7] in large autopsy series found the frequency of complete and incomplete duplication of the ureters to be from three to four per cent. Braasch and Scholl[8] cited 144 cases of

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duplication of renal pelves and ureters of which thirty per cent were complete and seventy per cent incomplete. Of these 144 cases there were only eight (5.5 per cent) in which bilateral duplication of pelves and ureters was present and all of these eight did not have four bladder orifices for the ureters. These authors also noted that 37.5 per cent of the cases presented some pathology of the urinary tract. Harpster, Brown and Delcher[9] in a review of the cases of duplication of the ureter in the literature up to 1922 found complete duplication in 58.1 per cent of the 382 cases reported up to that time. They recorded 181 instances of complete unilateral duplication, 40 of complete bilateral duplication, 133 of incomplete unilateral duplication, and 28 of incomplete bilateral duplication. They believe that only a very few cases of this condition are diagnosed pre-operatively, the difficulty being increased when the cystoscopic examination shows but two apparently normal ureteral openings in their usual positions.

From the number of cases of this form of anomaly of the urinary tract that are cited one might conclude that this condition should be encountered not infrequently in practice. That the diagnosis is seldom made is well known. The figures that have been quoted are derived from autopsy records or from observations made on a large series of operative cases. Mertz,[10] in 1920, compiled a list of the cases of duplicated ureters recorded to date. He came to the conclusion that many cases are discovered only post mortem and that of those diagnosed during life and before operation the diagnosis was possible only after *repeated* urological and x-ray examinations.

My purpose in here citing another case of bilateral double kidney and ureters is twofold: first, to point out that with the aid of newer laboratory methods this condition should be diagnosed more frequently; second, to show that the responsibility for this diagnosis need be borne no longer solely by the urologist but that the responsibility rests upon internist and the general practitioner as well, with the aid of the roentgenologist.

Report of Case

Case: A single woman, a school teacher, age 52, came October 18, 1930, complaining of pain and discomfort in the left lower quadrant of the abdomen and in the left lumbar region, the latter radiating at times down the left thigh. The pain had been present for as long as the patient could remember but had recently become more annoying. Lately there had been frequency of urination; she noted that on urination there was accentuation of the pain of the left side of the abdomen and of the left thigh. For the last four years there had been epigastric distress after eating; during this interval the patient was frequently awakened from her sleep by migrainous headaches. There was nothing of significance in the previous history. The family history was interesting in that her mother died at the age of 45 years with carcinoma of the bladder and one sister has been told that she has calculi of the urinary bladder.

The patient was well developed and very well nourished. Her height was 5 feet 1½ inches and her weight was 161 pounds. There was a systolic murmur over the apex of the heart which was considered as functional in nature. There was marked tenderness to palpation below the right costal border over the area of the gall bladder. No lumbar tenderness was noted. Other features of the physical examination were essentially normal. The blood pressure was 122/90. A tentative diagnosis of gall bladder disease and nephrolithiasis was made.

The patient had 4,220,000 erythrocytes and 6,700 leukocytes. The differential count showed 60 per cent polymorphonuclears, 2 per cent eosinophiles, 2 per cent large lymphocytes, and 36 per cent small lymphocytes. The hemoglobin was 90 per cent (Sahli). Repeated examinations of catheterized urine showed a few white blood cells and numerous red blood cells.

Cholecystography (after the oral administration of tetra-iodo-phenolphthalein-sodium) showed a gall bladder that filled satisfactorily and was reported as normal in size and shape and regular in contour. In the fundus of the gall bladder there was a small circular filling defect about the periphery of which there was a thin dense shadow. This concentric rim was visible two hours after the gall bladder had been emptied of the dye by the ingestion of a fat-full, mixed meal. A report of cholelithiasis was made.

It was considered unlikely that the biliary calculus was accountable for the left sided pain. Due to the persistent presence of blood cells in the urine the diagnosis of renal calculus was further advanced. The kidneys were x-rayed and there was no evidence of calculi. Both kidneys were reported as being considerably larger than usual. The patient was then given 40 grams of Uroselectan (sodium salt of 2-oxo-5-iodopyridine-N-acetic acid) intravenously. Twenty minutes after the intravenous injection of the dye there was a very clear visualization of two renal pelves on each side from each of which a ureter came off (figure 1). On both sides the more superior pelvis consisted of but a single calyx while the inferior and larger pelvis had two major calyces. A moderate degree of hydronephrosis of the two lower pelves was reported, more pronounced on the left side. On each side the ureter from the superior pelvis was situated medial to the one from the inferior pelvis. A picture made forty-five minutes after the dye had been injected gave no additional information. In the picture made sixty minutes after the dye had been given (figure 2) the ureters in their lower course are well seen. On the left side two distinct shadows placed very close together are noted down to the level of the symphysis pubis. Below this a single wider shadow is noted entering the bladder. It could not be ascertained definitely from the picture whether this represented a union of the duplicated ureters or whether it was due to overshadowing of the separate ureters. Although the right side did not show this condition so clearly, a similar arrangement could be made out on very close inspection of the films. The patient was referred for cystoscopic examination, but inasmuch as she refused this examination it could not be definitely made out whether two, three, or four bladder orifices were present.

Papin and Eisendrath[11] pointed out that the ureter belonging to the upper pelvis always ends lower and more mediad. In addition, they state that when there are two ureters from one kidney there are always two pelves on that side—no case has ever been shown to be otherwise. Bugbee and Losee[12] held that the presence of a double ureter means the existence not only of two pelves but also of two physiologically separate kidneys on the side involved, although these may be fused anatomically to represent a single organ. Braasch and Scholl[8] were of the opinion that hydronephrosis is the most common pathological complication of duplication of the ureters. They believed that this is most probably due to the fact that there is ureteral obstruction in the region of the junction of the two ureters in incomplete duplication. Harris[13] was of the same opinion and added that there is usually nothing in the history or physical examination to lead one to suspect anomalies. Thompson[3] found that the combined capacity of the pelves in double kidney may be less than that of a single normal pelvis. In such cases the gradual constriction of the normal pelvis may be replaced by a very sharp one, making the discharge of urine

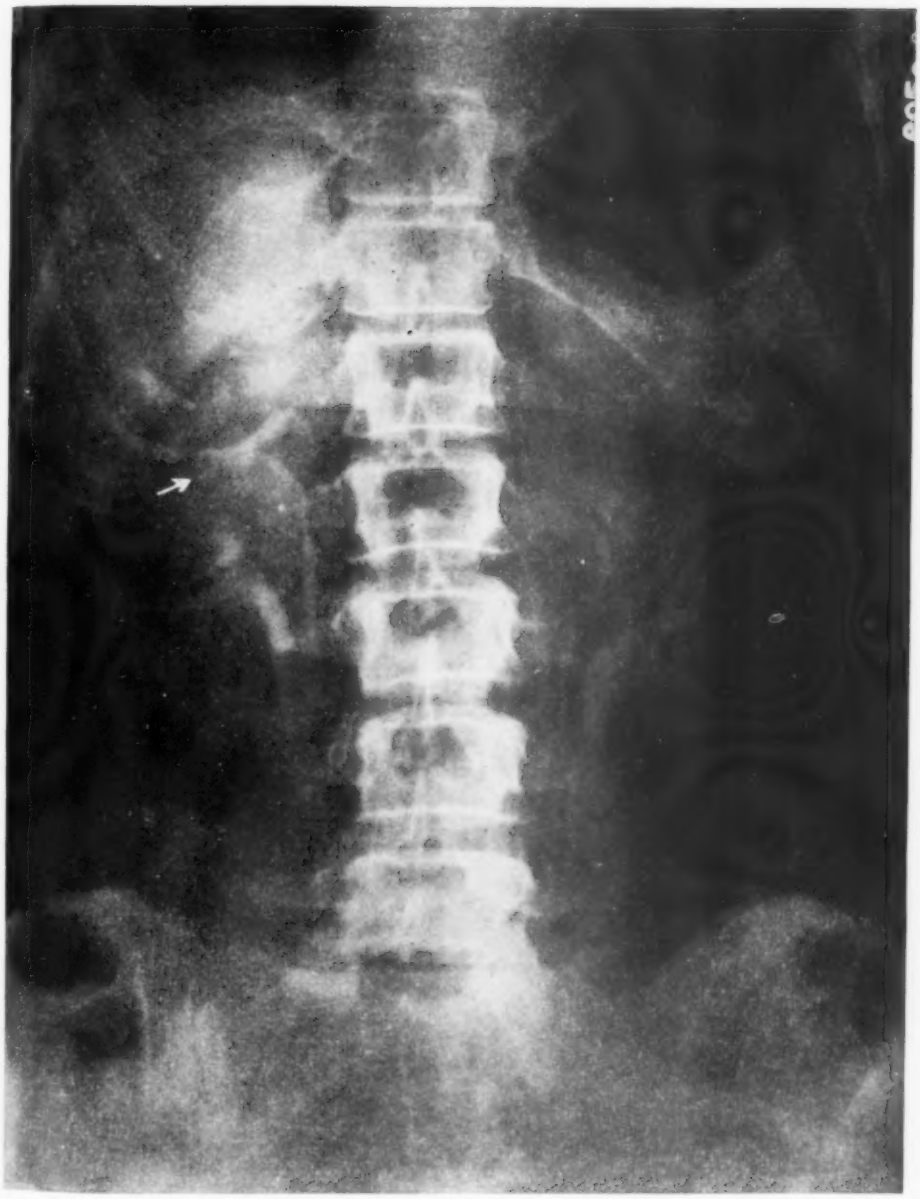

FIG. 1. Twenty minutes after the intravenous injection of the dye. The arrow indicates the biliary calculus; this might easily be mistaken for a renal calculus if cholecystography had not been successful.

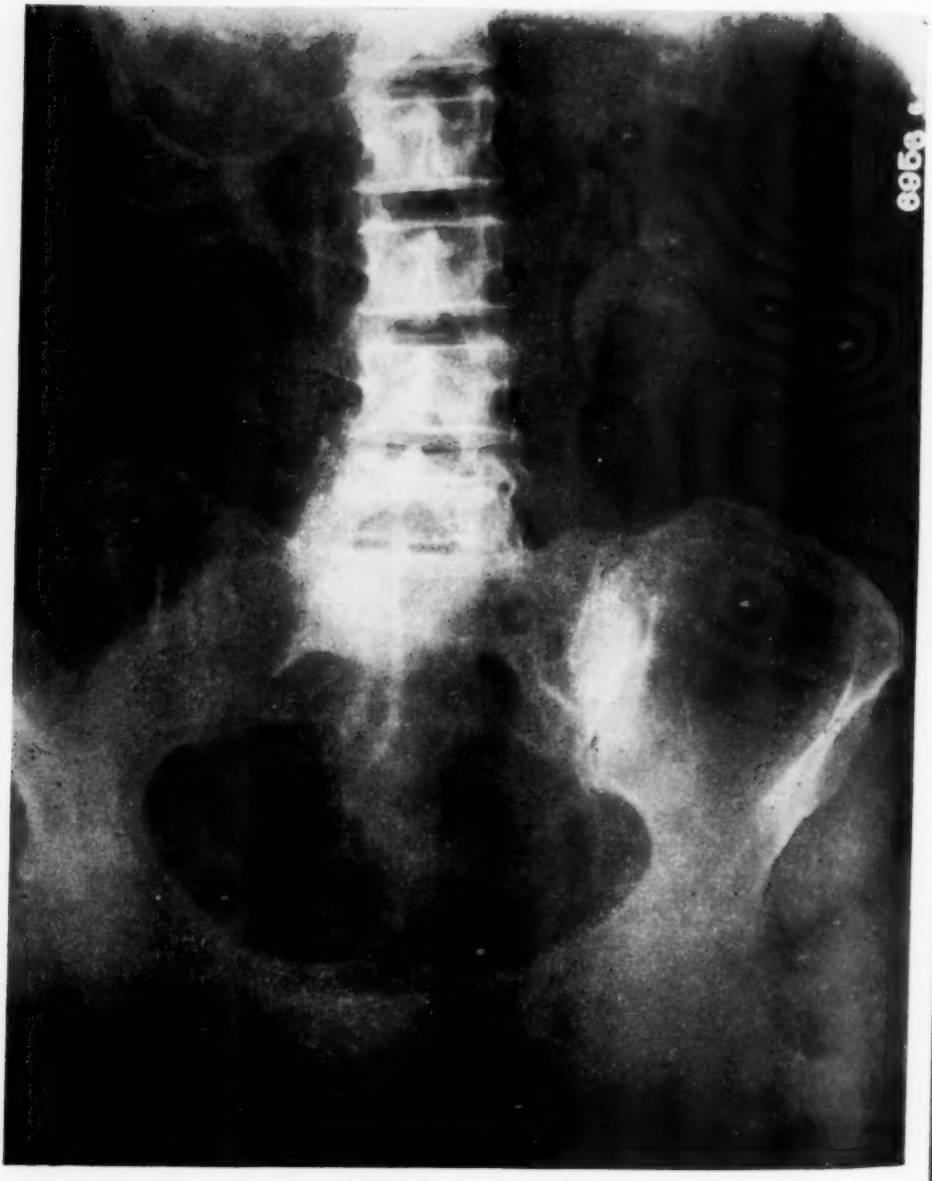

FIG. 2. Sixty minutes after injection of the dye. On the left side the course of the lower part of the ureters is clearly seen.

more difficult. He believes that at times pain and slight pyuria can be explained only on the basis of double small pelves with reduplication of ureters.

Embryology

The following brief summary of the embryological development of the urinary tract is taken mainly from the article of Young and Davis.[2] During the embryological development of the higher vertebrates there are three successive types of excretory organs. These are the pronephros, the mesonephros, and the metanephros—the latter becoming the permanent kidney. All three types come from the mesodermal blocks known as the nephrotomes. The nephrotomes consist of a series of blocks of mesodermal cells situated longitudinally along both sides of the neural canal and are between the primitive segments and the lateral mesodermal plates.

The Wolffian duct—the excretory duct of the mesonephros—is formed from the pronephros. The mesonephros arises from the nephrotomes that extend from the fifth cervical to the third lumbar segments and consists of a series of glomeruli and tubules opening into the common duct. In man this structure atrophies. According to Kelly and Burnam[14] the mesonephros is at the height of its development during the fourth and fifth weeks of embryonic life. The mesonephros atrophies during the eighth to sixteenth embryonic weeks, the Wolffian duct persisting as the vas deferens in the male and as the rudimentary Gartner's duct in the female.

The Wolffian duct opens into the cloaca and it is near this juncture that a budding occurs which is the anlage of the permanent kidney. From this bud is formed the ureter, pelvis, calyces, and collecting tubules while the secreting portion of the kidney is formed from a collection of mesodermal cells known as the metanephrogenic tissue. This latter mass of tissue surrounds the tip of the ureter bud soon after its formation.

The ureteral bud, capped with metanephrogenic tissue, first grows dorsally toward the spine and then turns cranialward. The tip of the bud at the 6.6 mm. embryo stage has become bulbous and as early as 8 mm. a bifurcation of the primitive pelvis occurs, the first evidence of the calyces. During the ascent of the kidney the uro-rectal septum appears, dividing the cloaca into what is to be rectum and bladder. Young and Davis believe that in double ureter the cause may be ascribed to premature or exaggerated bifurcation of the ureteral bud, the split extending varying distances down the ureteral stalk instead of being confined to the primitive pelvis.

Comment

The frequency of the occurrence of double kidney with duplication of the ureter and the very infrequent diagnosis made of this condition suggests that there is generally a nearly complete absence of symptoms that might be termed characteristic. When a patient does give a story that might be referable to the urinary tract, such an anomaly is rarely considered. With the aid of cystoscopic examination the frequency of diagnosis of double ureter is somewhat increased, but still the possibility of missed diagnosis is too great.

Assuming that retrograde pyelography is employed it is usual to visualize but one ureter where there is incomplete duplication. A reflux of the contrast medium into the lower portion of the accompanying ureter is usually the first hint of duplication of the ureter.

Where a history and physical examination are suggestive of an indefinite disturbance of the urinary tract the physician should consider the possibility of double ureter. With the introduction of intravenous pyelography the diagnosis becomes quite easy and very certain. This method, as is well known, is easy of application. The certainty with which the diagnosis is made or ruled out in kidneys of good function is apparent. Employment of this method makes for a more comfortable and satisfied patient. Where intravenous pyelography is used it may make cystoscopic examination unnecessary—a procedure to which many patients object both before and after its accomplishment.

It is to be expected that the preoperative diagnosis of double kidney and double ureter will be more frequent. The presence of this condition can usually be determined very satisfactorily by the internist or the general practitioner with the aid of an x-ray study.

Conclusions

1. The occurrence of double kidney with duplication of ureters and kidney pelves is not rare, but the diagnosis is seldom made.

2. Pain in the lower quadrant of the abdomen and in the lumbar region of the same side radiating down the thigh, frequency of urination, red blood cells and white blood cells in the urine, should lead one to consider the possibility of this anomaly after renal calculi have been ruled out.

3. Duplication of ureters and pelves is due to an abnormal exaggeration of the usual embryonal development.

4. Intravenous pyelography enables easier and more certain diagnosis of double ureters and pelves than does cystoscopy.

5. With proper x-ray studies the internist and the general practitioner should make the diagnosis of this condition.

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On the Treatment of Angina Pectoris

"THE spiritual side of the case must not be neglected in this disease in which the emotions play so imporant a rôle. The development of a philosophy of life, of the power of adaptation of desire to possibilities, the cultivation of hobbies of a restful character, such as suitable reading, music and such pacific occupations as painting, etching, carving and similar pursuits, are of real medical benefit. Habits of restfulness and relaxation are to be cultivated. Climate is often very important, and those who live in the temperate zones may well spend their winters in the South or perhaps go to live permanently in some mild, temperate and congenial climate. Few patients do well in the cold places or at high altitudes."—(Harlow Brooks, M.D., F.A.C.P., *Am. Jr. Med. Sci.*, 1931, clxxxii, 784-800.)

Editorial

INTERNAL RADIATION IN THE CAUSATION OF MALIGNANCY

A few months ago reference was made editorially in the ANNALS to the known extrinsic factors having a carcinogenic or sarcogenic action. X-ray and other solar irradiation have both been recognized for many years as belonging in this group. That the radio-active source may be within the body of the victim, inseparable from him, diminishing in amount and activity only by its natural and unalterable decay, and at the same time be capable of inducing neoplastic proliferation in a startling conception. Martland[1] has demonstrated that this is exactly the situation which obtains with certain of the radium dial painters. Knowledge of this fact has a significance much wider than its application to this extraordinary occupational disease. It may explain the extrinsic factor in certain other forms of occupational malignancy and sounds a warning in connection with the indiscriminate internal use of sources of radio-activity.

The circumstances under which radium poisoning was recognized as an occupational disease are now well known. In a New Jersey factory there were employed for varying periods of time during the years 1917 to 1924, about 800 girls whose work was painting the luminous dials of watches and clocks. The paint consisted of phosphorescent zinc sulphide combined with small amounts of radium, mesothorium and radiothorium in the form of insoluble sulphates. Due to the habit of pointing the brushes used between the lips, small amounts of radio-active paint were ingested over extended periods. The possibility of absorption through the skin and by inhalation also existed. Subsequent investigations have shown that, while most of the paint ingested passed rapidly through the alimentary tract and was eliminated, a certain portion was stored in the body and particularly in the bones. After final deposition in the bones these radio-active substances emit their characteristic radiations continuously, diminishing only with the exhaustion of the deposit by the natural process of physical-chemical decay, a process so slow in the case of radium that a life-time makes no significant change. It has been found from material obtained at autopsy that the lethal amount of radio-active substances distributed in the entire skeleton may range from 10 to 180 micrograms, estimated as radium element. As Martland vividly states it, it is necessary to have only 10 micrograms (one one-hundred-thousandth gram) of radium bromide, distributed over the entire skeleton, to

[1] MARTLAND, HARRISON S.: The occurrence of malignancy in radio-active persons, Am. Jr. of Cancer, 1931, xv, 2435-2516.

produce a horrible death years after it has been ingested. The damage in the radium dial painters is due to the internal bombardment with alpha particles, a type of radiation never before known to have occurred in human beings.

Martland has knowledge of 18 deaths, among the former employees of this factory, which can be strongly suspected of being due to so-called radium poisoning. Eight were proved to be such by autopsy. In addition, there are about 30 persons alive who are suffering from typical symptoms of radium poisoning or who, by virtue of their internal radio-activity, are liable to develop crippling lesions at any time. During the first six years after the development of this industry the ill-effects and resulting fatalities fell into two chief groups. One of these was characterized by a radiation osteitis, with a superadded bacterial infection in the case of the mandible and maxilla, so that extensive necrosis of the jaw preceded the fatal outcome. In the other group the continuous bombardment of the bone marrow resulted in a regenerative type of leukopenic anemia which resisted all efforts at treatment. During the years of this earlier period, that is, from 1922 to 1928, two dial painters died from osteogenic sarcomas. These were reported in 1929 by Martland and Humphries[2] who recognized that the incidence of two cases of sarcoma among fifteen examples of radium-mesothorium poisoning was too large to be passed over as due to coincidence. This belief has now been fully justified for Martland's more recent report records the results of autopsy studies upon three more fatal cases of anaplastic osteogenic sarcoma, all of the victims coming from the group who worked in the New Jersey factory. In addition, he lists three additional living cases of probable osteogenic sarcoma from the New Jersey group and another fatal case in a girl who had worked as a dial painter in factories in other states.

In view of the known incidence of primary bone sarcoma in the general population, the occurrence of so many examples in the limited group exposed to this occupational hazard can have but one explanation—internal radiation has operated as a causal factor. Actual malignancy has been preceded by radiation osteitis, associated at first with a hyperplastic, irritative, compensatory bone marrow which is succeeded by a replacement fibrosis. The aplastic marrow of external irradiation is not found.

Knowledge of an occupational hazard should lead to its mitigation and eventual elimination. The demonstration of this sarcogenic agent has a wider significance, however, than its importance in the dial-painting industry. It affords a basis for a high degree of certainty that the occupational primary carcinoma of the lungs, which has been the most common cause of death among the cobalt miners of the Schneeberg district in Saxony for five hundred years, is best explained by the radio-activity of the ore. The air of these mines has a radio-active ema-

[2]Martland, Harrison S., and Humphries, Robert E.: Osteogenic sarcoma in dial painters using luminous paint, Arch. Path., 1929, vii, 406-417.

nation content of from a few to 50 Maché units. A high incidence of primary carcinoma of the lung is said to have appeared among the workers in the pitchblende mines of Joachimsthal. It must be accepted that the deposit of radio-active substances in the body, particularly those producing alpha rays, may give rise to malignancy many years afterwards. From this, it follows that the indiscriminate lay and quack use of radio-active waters, emanators, activators, etc., should be stopped. They are without therapeutic value aside from their psychic effect and may be doing an, as yet, unrecognized harm. It may be learned that even slight increase in the normal radio-activity of the human body is not without danger.

San Francisco—The City by the Golden Gate

SINCE the American College of Physicians will hold its Sixteenth Annual Clinical Session in San Francisco, April 4 to 8, 1932, it is appropriate to call attention at this time to certain facts of general and historical interest about the city in which we are to gather.

San Francisco is not only one of the world's most beautiful and fascinating cities, it is also the center from which nine-tenths of California's scenic wonders can be most easily reached. Within a few hours' easy journey by train, stage or car are most of the places which tourists come thousands of miles to see. Yosemite lies due east, and can be easily reached in seven hours by automobile or train. Monterey Peninsula—one of the finest strips of sea coast in the world—is three and one-half hours by train to the south. The giant redwoods begin right in San Francisco's suburbs, at Muir Woods, and can be visited in half a day's excursion.

Lake Tahoe, at the summit of the Sierra, is an easy night's or day's run by train or a short detour *en route* or returning. Shasta, Lassen, Sequoia National Parks, the Redwood Empire, the wine-grape valleys, Stanford and California Universities, Luther Burbank's gardens, Jack London's Valley of the Moon—all are within the circle of San Francisco excursions, requiring from a few hours to a day or two.

One of the World's Truly Cosmopolitan Cities

San Francisco is famous for its gay spirit, its Chinatown, its beaches, its picturesque waterfront, its flavor of Asia and the sea, its French and Italian restaurants, its Parisian touch. Not so many realize that it is also the business capital of the West and a seaport

San Francisco's skyline from the Bay.

Cosmopolitan scene.

known and loved in many a city of Asia and Central America and the islands of the Pacific. Here is the financial center of the Coast, the home of the great corporations that dominate Western business, the seat of banking, insurance, commercial and industrial enterprises that function from Chile to Alaska and from Salt Lake City to Shanghai and Singapore.

A Town to Play In

From the days of the Gold Rush, San Francisco has believed in laughter and good living. People on the streets are happy and cordial. They smile easily. Good feeling and high spirits are in the air. You can't feel downhearted when you breathe the city's bracing sea-tang and see its hill rising in the sparkling sun above the blue Bay.

For scenic beauty, few places in the world can excel San Francisco itself.

Fisherman's Wharf.

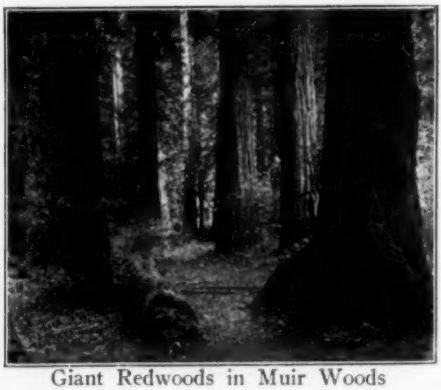
A tour of the city can be made as exciting and as filled with surprises as a first visit to some picturesque foreign town. The Presidio military reservation, founded by the Spanish in 1776, is one of the largest and most beautiful army posts in the country. Golden Gate Park is known around the world. The Ocean Beach and the Cliff House with its seal rocks, the public golf courses of Lincoln Park overlooking the Golden Gate, the eminences of Russian and Telegraph Hills, the quaint cable cars on the steeper streets, the picturesque foreign quarters, the glowing flower stands lining the curbs in the shopping district—all of these provide endless entertainment and diversion.

Montgomery Street, San Francisco.

Lure of the Ships

Business and cultural metropolis of the Pacific Coast, San Francisco is first of all a great sea-port. In scenic grandeur, its great land-locked harbor ranks with Rio, Naples, and Constantinople. To visit the waterfront, only a few minutes from the hotel and business section, is to feel one's self in touch with many strange far-off lands. You will see ships arrive and depart from Asia and the South Seas, for Latin America and Europe. You will hear strange tongues, see strange faces, smell the spice and fruits of the trop-

Market Street, San Francisco.



Giant Redwoods in Muir Woods

ics, watch endless bales of raw silk swung from the hold of a great trans-Pacific liner. San Francisco is the gateway to the Hawaiian Islands, with frequent sailings via famous *de luxe* liners. San Francisco is the center from which American travelers, American ideas, and American goods are carried to the hundreds of millions of awakening peoples inhabiting the great Pacific basin. Here the Orient and South Seas discharge their treasures. On these docks spill copra from the South Seas and Philippines, silks and teas from Japan and China, coffee and bananas from Central America, peasant ware from Spain and Italy.

Pacific Area's Treasure-House of Art

For eighty years the people of the Western Slope and of the Pacific Islands have looked to San Francisco for entertainment and instruction. Today traveling Europeans are amazed by the treasures of its galleries, the excellence of its great orchestra, the vitality and stir of its cultural life. The Palace of the Legion of Honor, a

Mission Dolores, founded 1776.

beautiful white marble replica of the famous Paris original, occupying a magnificant site commanding the Golden Gate and the open Pacific, holds priceless collections. So does the M. H. deYoung Memorial Museum in Golden Gate Park, and the beautiful California School of Fine Arts on Russian Hill. San Francisco has always been beloved by the stage, and its many theatres offer a variety of fare throughout the year.

Memorial Chapel at Stanford University.

You Can Golf, Fish or Swim

Within twenty minutes of your hotel are golf courses where Bobby Jones could drive a ball from a putting-green high on the cliff right into the Pacific Ocean. Not even old Edinburgh has such a setting for its national game. You will be welcomed to the city's finest courses. Perhaps you would rather board a sturdy launch and fish for sea-bass or salmon in the Bay, or outside the Heads in the open Pacific. It is easily arranged. So is trout-fishing in the Sierra or Coast-Range streams. For swimmers, there are the ocean beach, the famous in-door Sutro baths, and the Fleishhacker municipal open-air pool, the largest in the world.

You'll Feel at Home.

The City is a paradise for those who enjoy good food. There are lit-

Memorial Stadium at University of California, Berkeley.

Lincoln Park Municipal Golf Course overlooking the Golden Gate.

DeYoung Memorial Museum, Golden Gate Park.

erally thousands of restaurants and their rates are surprisingly low. Food is important in San Francisco. Restaurateurs are artists as well as business men. Many are well-loved town characters. You feel the difference. And you like the friendliness. It's a happy town. Around the corner from the big hotels are Italian and French restaurants famous for a soup, a salad, a specially-prepared sea-food served with care and pride at low prices. You can dine in China, Russia, Japan, Sweden, France, Spain, Italy or Germany by merely calling a taxi or walking a few blocks through streets rich in reminders that this is a great cosmopolitan sea-port. For five cents you ride to the Beach, and lunch or dine with surf pounding outside and nothing between you and Asia except the Pacific and a few islands. Or you can mount Telegraph Hill, rising steep between the docks and the Latin Quarter, and sit at tables commanding a superb expanse of blue salt water and encircling mountains. San Franciscans themselves like to go to Fisherman's Wharf, where the Madonna-blue boats bob with the tide, and dine in Italy on fresh-caught sea-food. The Bay, the Golden Gate, and Mt. Tamalpais look on you there.

Chinatown

San Francisco's Chinatown is world famous. Here are more dazzling displays of objects of art, silks, china and curious knick-knacks than are to be found in cities of China itself. Here

San Francisco's world famed Chinatown.

also are the two largest Chinese theaters outside of China, playing the weird dramas of Cathay with Chinese orchestras and every touch complete.

San Francisco's Distinctive Climate

In the summer it is America's coolest city — the warm California sun tempered by sea breezes and an opalescent reminder of sea mist which often takes the form, toward evening, of billowing white fog bringing the breath of the sea. Yet no circumstantial description of San Francisco can account for its peculiar appeal to the visitor without taking into account a certain atmosphere of romance and charm that is instantly felt and that proves invariably winning to the stranger. It is compounded of the unusual physical setting and climate, of the happy, healthy people, of the high hills and stunning vistas, of the foreign settlements and the oriental flavor, and also of a long tradition of stirring and romantic days, from the Spanish down through the Forty-niners to the bonanza kings of the Comstock Lode, the builders of our first transcontinental railroad, and the more recent generation that built a great modern city on the ruins of the old.

Mt. Tamalpais.

Abstracts

A Study of 503 Cases of Pulmonary Tuberculosis with Indefinite or No Usual Abnormal Physical Signs. By LAWRASON BROWN. (Am. Jr. Med. Sci., 1931, clxxxii, 700-707.)

"As far back as the beginning of the century, Osler, among others, recognized that extensive disease of the lungs could exist with few or no abnormal physical signs. . . . I wish to present to you some conclusions I have arrived at from a study of some 503 such cases . . . selected from 1900 consecutive cases at the Trudeau Sanatorium . . . The method by which a diagnosis of chronic pulmonary tuberculosis was established in these cases is that used for some years at the Trudeau Sanatorium. The occurrence alone of either hemoptysis of a drachm or more, or of pleurisy with effusion, explainable on no other grounds, is considered to justify a diagnosis of suspected pulmonary tuberculosis in the absence of all other symptoms. . . . The occurrence of moderately coarse râles or of a parenchymatous roentgen ray lesion (mottling, irregularly distributed) above the third rib and third vertebral spine was considered sufficient evidence for a positive diagnosis of pulmonary tuberculosis until disproved. Tubercle bacilli in the sputum in the absence of lesions above the trachea must be considered as absolute proof of pulmonary tuberculosis. The incidence of these five cardinal diagnostic data in 1367 cases diagnosed pulmonary tuberculosis from 1478 consecutive cases admitted to the Trudeau Sanatorium is as follows:

	Per cent
Tubercle bacilli	61.5
Râles	68.5
Roentgen ray	99.0
Hemoptysis	33.5
Pleurisy	12.0

. . . From the data here presented I am inclined to attribute considerable importance in the diagnosis of pulmonary tuberculosis to the roentgen ray examination. I am convinced it will reveal the lesions of the disease long before it is manifested in any other way."

Viosterol Treatment in Experimental Hyperparathyroidism. By HENRY L. JAFFE, AARON BODANSKY and JOHN E. BLAIR. (Proc. Soc. for Exp. Biol. and Med., 1931, xxix, 207-208.)

Three groups of experimental animals (guinea pigs) were established. To those in the first group viosterol was given daily for 7 to 10 days. After this preliminary treatment, the animals were injected daily with parathormone in increasing amounts, and the viosterol was also continued. This combined medication extended over a period of 23 days. In a second group viosterol and parathormone were started simultaneously and administration extended over a period of 29 days. The third group contained 6 controls which were injected with parathormone as in the second group, but these animals did not receive viosterol. The animals in all three groups were killed to terminate the experiment and their bones were taken for histological examination. They all showed decalcification and secondary fibrous invasion of the bones. There was no consistent difference in the nature or severity of the lesions between the guinea pigs receiving viosterol and parathormone (groups 1 and 2) and those receiving only parathormone. Under the conditions of this experiment irradiated ergosterol did not protect from the demineralizing effects of experimental hyperparathyroidism. It is possible, however, that the healing of bone lesions in experimental or clinical hyperparathyroidism might be promoted by viosterol after the state of hyperparathyroidism had been terminated by discontinuance of parathormone administration, or by the removal of a parathyroid adenoma.

Nonsyphilitic Aortic Valve Deformity. By B. J. CLAWSON. (Arch. Path., 1931, xii, 889-899.)

Nonsyphilitic aortic valve disease is of two kinds: active vegetative endocarditis, and deformity due to thickening and roughening with scar tissue and often with calcium deposits. The frequency, and particularly the etiology, of the latter type have been subjects of much discussion in the last few years. The author's series contains 93 cases of aortic valve deformity of the calcified nodular type. In 91 per cent of 68 cases in which this point was investigated, some degree of stenosis was present. It is evident that aortic stenosis of a grade recognizable clinically, and due to this type of valvular deformity, is far more rare. Both gross and microscopical findings in these valves tend to support the view that the etiological factor is an inflammatory rather than a metabolic (atherosclerotic) condition. The frequency of rheumatism in these cases, as indicated by a previous positive history, by the presence of an adherent pericardium or by an association of deformities in other valves, strongly supports the view of an infectious basis. It is doubtful whether a valve deformity severe enough to cause cardiac insufficiency is ever due to a metabolic disturbance such as arteriosclerosis. Accordingly the term 'arteriosclerosis valve deformity' should not be used in describing valvular insufficiency or stenosis.

Effect of Giving Digitalis on the Volume Output of the Heart and its Size in Heart Failure. By HAROLD J. STEWART. (Proc. Soc. for Exp. Biol. and Med., 1931, xxix, 207-208.)

The method of Grollmann for measuring the cardiac output was utilized in studying the effect of digitalis on the heart of normal individuals. Digitan (Merck) was given in a single dose of 0.8 gm. to 1.0 gm. Observations were made immediately before the drug was given and at frequent intervals afterward. All observations were made with the subjects in a basal metabolic state. In addition to measurements of cardiac output, the cardiac size was measured on x-ray photographs made at a distance of two meters. Electrocardiograms were made also; the heart rate was counted and the blood pressure recorded. Consistent results were obtained from the four subjects studied. With the administration of digitalis to normal men (1) cardiac output *decreased;* (2) the cardiac size *decreased;* (3) the cardiac rate *decreased;* (4) alterations of the T-wave of the electrocardiogram occurred; (5) the blood pressure was usually elevated; (6) the maximum effects were observed 9 to 24 hours after the drug had been given, and had usually passed off at the end of 48 hours.

Effect of Giving Digitalis on the Volume Output of the Heart and its Size in Heart Failure. By HAROLD J. STEWART. (Proc. Soc. for Exp. Biol. and Med., 1931, xxix, 209-211.)

The methods referred to in the preceding abstract were used in a study of the effect of digitalis on the volume output and size of the heart in heart failure. A small group of patients, among whom were included examples of arteriosclerotic, luetic and rheumatic heart disease, with and without disturbances of rhythm, gave results indicating that in the presence of heart failure (1) the cardiac output diminishes. (2) With the administration of digitalis (a) the cardiac output increases; (b) the cardiac size diminishes; (c) the ventricular rate decreases both when the rhythm is normal and in the presence of auricular fibrillation; (d) and alterations in the form of the T-wave of the electrocardiogram occur. (3) As the effect of digitalis wears off, these functions change in the reverse direction.

Tularemic Leptomeningitis. By ARTHUR R. BRYANT and EDWIN F. HIRSCH. (Arch. Path., 1931, xii, 917-923.)

A chef, aged 48, died on the sixteenth day after lacerating a finger with a rabbit bone while removing *pickled* rabbit from a jar. At autopsy focal lesions were found in the leptomeninges, contiguous brain tissues, ependyma, subependymal tissues and choroid plexus, which were similar to lesions in the liver, spleen and lungs. All of these had the characteristics of tularemic lesions. In addition there was a diffuse acute exudative meningitis. The serum of the patient, taken

on the fourteenth day, was reported to agglutinate *Bacterium tularense* in dilutions not greater than 1:40. This low titer is not considered to militate against the diagnosis of tularemia when the serum was taken as early as the fourteenth day from a patient whose failure to form antibodies was indicated by his death two days later. Differentiation from miliary tuberculosis was made possible in that suspensions of the crushed lesions killed guinea pigs in five days and produced in them visceral lesions characteristic of tularemia, staining for tubercle bacilli gave only negative results, and lesions of a frankly tuberculous character were not found in the course of a thorough gross and histologic examination of the viscera of the patient.

Reviews

Medical Jurisprudence. By CARL SCHEFFEL, Ph.B., M.D., LL.B. xii + 313 pages. P. Blakiston's Son and Company, Inc., Philadelphia, 1931. Price, $2.50, postpaid.

Contrary to the procedure of most writers on this subject, Scheffel treats of the effect of law upon medical practice and practitioners rather than the reverse. The material presented is of a general character so that the book should be of service to the entire medical profession. There is no attempt to impart highly specialized knowledge applicable only to restricted medical groups. The major divisions of law as they affect the physician are discussed with particular emphasis upon what the author chooses to term "legal prophylaxis"; that is, the prevention of unfortunate legal complications involving the medical practitioner. The subjects treated include contractual relationships, law of agency, torts or civil wrongs other than breach of contract, principles of evidence, function of the medical witness, property interests of physicians, criminal responsibility and physicians as law makers. Much of value to the average practitioner is contained in the chapters on contracts and agency. The types of contracts occurring in medical practice, and the specific liabilities of physician and patient in each are well illustrated with specific examples, and many practical suggestions are made to aid in avoiding unnecessary liability. There is included an exposition of various types of commercial contracts which the medical man is especially prone to make, oftentimes unwittingly; herein is timely and practical advice which should be invaluable. The function of the physician in court is developed at length and an effort is made to show how this relationship affects both the process of justice and the physician himself. The rights of the witness, expert and otherwise, are outlined from the legal point of view, and the importance of an adequate comprehension of the strict meaning of terms commonly used in court is stressed. In general it may be said that the book reflects a legal training rather than a medical one; that it deals with the lawyer's view of the physician rather than with the latter's evaluation of his problems of law and social responsibility. This is particularly evident in the section dealing with autopsies wherein attention is called to a supposed disadvantage of the autopsy because it is thereby frequently shown that the clinical diagnosis was erroneous and that treatment had been applied for a condition that did not exist; thus the liability for malpractice suits may be increased. If such fears were to dominate the medical profession, then progress in scientific medicine would be practically nil. The emphasis on this aspect of the autopsy seems peculiarly ill-advised. Notwithstanding such points which may be called in question, this attitude of mind on the part of the author is in keeping with the purpose of the book and is undoubtedly a large factor in making the work of greater personal value to members of the medical profession than many similar publications. The manuscript is well organized, the argument is logical, the style vivid; only numerous typographical errors and the rather frequent use of a plural pronoun with a singular antecedent mar an otherwise artistically compiled treatise.

Clinical Dietetics: A Textbook for Physicians, Students and Dietitians. By HARRY GAUSS, M.S., M.D., F.A.C.P.; Instructor in Medicine, University of Colorado, School of Medicine; Assisted by E. V. GAUSS, B.A., Formerly Assistant Dietitian, Presbyterian Hospital, Denver, Colorado. 490 pages, 59 illustrations. The C. V. Mosby Co., St. Louis, Mo., 1931. Price $8.00.

The needs of three distinct groups, students of dietetics, medical students, and practitioners, were kept in mind as this textbook was being written. Certain difficulties are inherent in the attempt to present the subject matter of use to each of these groups without becoming tiresome to the others. Yet the authors seem to have succeeded to an unusual degree. The first four chapters give a brief but interesting historical outline, a survey of the nature of foods, of dietetic principles, and of the theory of digestion. The latter half of the book is concerned with clinical applications of dietetic principles. A few minor flaws—at least, they appear to be such to the reviewer—can be easily remedied in a second edition. It is unfortunate that vitamin E is made to include the pernicious anemia preventing principle, as well as the anti-sterility factor to which this letter should be restricted. Although the fact that both glomeruli and tubules frequently share in nephritis is clearly set forth in the text, the use of Richard Bauer's super-simplified diagram is unfortunate. There are also a number of small but important typographical errors which need correction. The use of historical material is skillfully managed and the introduction of case histories in smaller type adds much to the clinical interest and value. It is encouraging to those who stress in their teaching the importance of constitution, in the sense of Pende, to find that no less than 16 pages are given over to the significance of bodily habitus in matters of digestion and dietetics. That every patient is an individual whose special physiological, psychological and morphological peculiarities must be evaluated with understanding is frequently reiterated. A simple and pleasing style is one of the most important of the many good features of this book.

A Manual of Clinical Laboratory Methods. By CLYDE LOTTRIDGE CUMMER, Ph.B., M.D., F.A.C.P.; formerly Associate Clinical Professor of Clinical Pathology, School of Medicine, Western Reserve University, Cleveland; Instructor in Dermatology and Syphilology, School of Medicine, Western Reserve University; Visiting Dermatologist, Charity and St. Alexis Hospitals, Cleveland, Ohio. Third Edition, thoroughly revised. 585 pages, illustrated with 173 engravings and 12 plates. Lea and Febiger, Philadelphia, 1931. Price $6.75, net.

This manual of laboratory methods should be of value to the medical practitioner, as well as to the laboratory technician, because of its style of presentation, which, though concise, gives minute details of even simple procedures. The book consists of twelve chapters, the first five of which deal with the blood, its examination, differential diagnosis of blood dyscrasias, parasitology and bacteriology of the blood, immunology and blood chemistry. An appendix gives an efficient method for examining a large number of urine specimens in a hospital laboratory. This portion of the book also takes up the preparation of normal solutions, the preparation of stains and of autogenous vaccines, as well as other information of interest and value. The last two pages of the book are made up of a Table of Normal Findings, which will be appreciated particularly for the more unusual laboratory procedures. The author presents the newer tests as well as the recent modifications of the standard methods. He describes Kline's Macroscopic Slide Test for syphilis, commenting briefly upon its value. The charts and illustrations are, on the whole, very good and a distinct addition. A bibliography requiring 19 pages gives citations to the original articles in which the technical methods now in vogue were described.

Hypertension and Nephritis. By ARTHUR M. FISHBERG, M.D.; Associate Physician to Beth Israel Hospital; Adjunct Physician to Mount Sinai Hospital, New York City. Second edition, thoroughly revised and enlarged. xvi + 619 pages, 38 engravings and one colored plate. Lea and

Febiger, Philadelphia, 1931. Price, $6.50 net.

For this second edition of Hypertension and Nephritis the entire book had to be reset, so extensive was the revision. The sections on renal acidosis, azotemia with chloride deficiency, the Addis ratio, the pathogenesis of edema, the kidney in diabetes and hemoglobinemia, the rôle of sensitization in glomerulo-nephritis, renal osteo-dystrophy, the carotid sinus and regulation of blood pressure, cardiac failure in hypertension, and paroxysmal hypertension with suprarenal tumors are either new or have been rewritten. As a result, in this edition the number of pages has been increased by about 50. This is a very complete and satisfactory discussion of the subject, written with the needs of the general practitioner especially in mind. The simpler methods of clinical investigation are stressed and their value emphasized. Throughout management and treatment are set forth in connection with the discussion of each clinical group. This practical clinical application does not mean that the method suffers from lack of scientific analysis, for both morphological pathology and altered physiology are thoroughly presented. Not all pathologists will agree with the elimination of the tubular system from the conception of a defensive nephritis, but approval will be general for the elimination of the term chronic interstitial nephritis, and the firm stand that there is no justification for its use "as commonly applied to those renal diseases which are characterized clinically by arterial hypertension and its consequences."

A Text-Book of Pathology. By FRANCIS DELAFIELD, M.D., LL.D., sometime Professor of the Practice of Medicine, College of Physicians and Surgeons, Columbia University, New York City; and T. MITCHELL PRUDDEN, M.D., LL.D., sometime Professor of Pathology, College of Physicians and Surgeons, Columbia University, New York City. Fifteenth edition, revised by FRANCIS CARTER WOOD, M.D., Director of the Pathological Department, St. Luke's Hospital, New York; Director of the Institute of Cancer Research, Columbia University, New York. 1339 pages, 20 full-page plates, 830 text illustrations. William Wood and Company, New York City, 1931. Price, $10.00.

No extended review of this well and favorably known textbook is needed. The present edition, the fifteenth, appears after an interval of four years during which time many notable advances in the field of general pathology have been made. Those additions which appear significant and lasting have been added in the revision. The task of including within one book the essentials of both general and special pathology grows more difficult of accomplishment with each succeeding year. Detailed treatment of any one field under such circumstances has become entirely impossible. This well-balanced text continues to be one of the most satisfactory sources for the fundamentals of pathology to be found in the English language. The addition of references to recent reviews directs the reader to more extended information. Many more citations could have been made to advantage.

College News Notes

Acknowledgement is made of the receipt of gifts to the College Library of publications by members, as follows:

Dr. Clarence L. Andrews (Fellow), Atlantic City, N. J.—1 book, "How's your blood pressure";

Dr. William W. Cadbury (Fellow), Canton, China—5 reprints;

Dr. Nathan S. Davis, III (Fellow), Chicago, Ill.—4 reprints;

Dr. George H. Lathrope (Fellow), Newark, N. J.—8 reprints;

Dr. Eugene P. Marzullo (Associate), Brooklyn, N. Y.—1 reprint;

Dr. Aaron E. Parsonnet (Fellow), Newark, N. J.— 1 reprint;

Dr. Martin J. Synnott (Fellow), Montclair, N. J.—1 reprint;

Dr. Fritz B. Talbot (Fellow), Boston, Mass.—1 reprint;

Thorndike Memorial Laboratory, Boston, Mass.—41 reprints.

Dr. Henry Daspit (Fellow), New Orleans, Louisiana, Dean of the Graduate School of Medicine of The Tulane University of Louisiana, participated November 23 and 24 in a Medical Institute at Rutherford Hospital, Murfreesboro, Tennessee, presenting papers entitled "Epidemic Encephalitis in General Practice" and "Preventive Medicine Aspects of Psychiatry", and giving a neuro-psychiatric clinic.

Dr. Harold F. Machlan (Fellow), has been transferred from the U. S. Veterans' Hospital, Lake City, Florida, to become Clinical Director of the U. S. Veterans' Administration Hospital at Indianapolis, Indiana.

Dr. Roy C. Mitchell (Fellow), Mount Airy, North Carolina, addressed the Grayson-Carroll County Medical Society of Virginia, November 9, on the subject, "Indigestion After Forty".

Dr. Wade W. Oliver (Fellow), Professor of Bacteriology in the Long Island College of Medicine, Brooklyn, N. Y., with his wife, recently returned from an eighteen months' trip around the world. Dr. Oliver was sent by the Rockefeller Foundation as Visiting Professor of Bacteriology and Immunology in the School of Hygiene and Public Health of the University of the Philippines, Manila. He served there during the teaching year of ten months from May, 1930, until March, 1931. The remaining eight months were spent in visiting bacteriological laboratories in, and touring Hawaii, Japan, Korea, China, Bali, Java, Federated Malay States, Siam, Burma, India, north Egypt, Italy, France and Germany.

Dr. Oliver T. Osborne (Fellow), New Haven, Conn., is the author of an article entitled, "Foods and Health", which appeared in the Illinois Dental Journal for November, 1931. Dr. Osborne is also the author of an article in the Stomatologic Record, New York City, September-October, 1931, entitled "My Viewpoint of Dentistry".

Dr. Carl R. Howson (Fellow), of Los Angeles, California, was recently elected President of the Southern California Medical Association. Dr. Harry Henderson (Fellow), Santa Barbara, was elected First Vice President, and Dr. Robert Ramsay (Fellow), Pasadena, was elected Second Vice President. Dr. Fred B. Clarke (Fellow), Long Beach, was the retiring President.

Dr. H. Beckett Lang (Fellow), was recently appointed Director of Clinical

Psychiatry at the Marcy State Hospital, Marcy, Pennsylvania.

Dr. Lang addressed the Fulton Medical Society, October 20, 1931, on "The Mental Hygiene Clinic in the Community". On the same date he also addressed the District Health Officers at Syracuse on "The Malarial Therapy in Neurosyphilis". On November 2, 1931, Dr. Lang addressed the Parent Teachers Association of Hamilton, New York, on "Behavior Problems and Their Relation to Mental Illness".

Dr. George L. Pinney (Fellow), Hastings, Nebraska, was recently elected Secretary and Treasurer of the Republican Valley Medical Society. Dr. Pinney recently addressed the Nebraska State School Teachers Association on "Common Sense Pointers on Physical Education".

Dr. Clough Turrill Burnett (Fellow), Associate Professor of Medicine at the University of Colorado School of Medicine, delivered addresses at the Colorado State Medical Society in September, 1931, and at the Boulder County Medical Society in Longmont on November 12.

Dr. George H. Hoxie (Fellow), Kansas City, Missouri, was elected President of the Missouri State Tuberculosis Association at its last meeting in St. Joseph, during October. Dr. Hoxie is Medical Director of the Open Air Schools in Kansas City, as well as of the Kansas City Tuberculosis Society.

The following Fellows of the College were on the program of the Tenth Annual Meeting of the Eastern Homeopathic Medical Association held at Trenton, New Jersey, November 4, 5, and 6: Dr. Donald R. Ferguson, Philadelphia; Dr. E. Roland Snader, Jr., Philadelphia; Dr. G. Harlan Wells, Philadelphia; Dr. Milton J. Raisbeck, New York; and Dr. Linn J. Boyd, New York.

Dr. Lewis Jefferson Moorman (Fellow), Dean of the University of Oklahoma School of Medicine, Oklahoma City, is the President-Elect of the Southern Medical Association, having been elected at the recent meeting in New Orleans.

Dr. Frank Smithies (Master), Chicago, was elected President of the American Society of Tropical Medicine at its Twenty-Third Annual Session, held at New Orleans, November 19, 1931.

Dr. Albert E. Russell (Fellow), of the U. S. Public Health Service, Washington, D. C., delivered an address on Silicosis and Tuberculosis before the Milwaukee Academy of Medicine, Milwaukee, Wisconsin, on November 10, 1931. Dr. Russell also read a paper on Occupation and Respiratory Diseases before the Section on Public Health of the Southern Medical Association in New Orleans, November 20, 1931.

Dr. Karl D. Figley (Fellow), Toledo, Ohio, presented a paper on "Food Allergy" before the Academy of Medicine of Toledo and Lucas County, November 6, 1931.

Dr. Samuel M. Feinberg (Fellow), Chicago, addressed the Rock Island County (Illinois) Medical Society on "Allergy in General Practice", December 8, 1931.

Dr. Edgar Mayer (Fellow), Saranac Lake, New York, Associate Professor of Medicine, New York Post-Graduate Medical School and Instructor at the Trudeau School of Tuberculosis, was the guest speaker at a special meeting of the Pacific Physical Therapy Association, held at Los Angeles, December 9, 1931. Dr. Mayer's subject was "Are We Abusing our Patients with Light".

Dr. Henry M. Moses (Fellow), Brooklyn, New York, addressed the Medical Society of the County of Kings, November 17, 1931, on "Pulmonary Neoplasms—The Clinical Findings and Methods used in Diagnosis".

Dr. Franklin B. Bogart (Fellow), Chattanooga, Tennessee, read a paper before the Radiological Section of the Southern Medical Association, at New Orleans, in November, on the subject "X-Ray Examination of the Heart in Left Auricular Enlargement".

Dr. Hyman I. Goldstein (Associate), Camden, New Jersey, during September and October, 1931, addressed the Krankenkassa physicians in Vienna on "The Physician in American Life To-day—His Position; Socially, Economically, and Scientifically". He spoke on the subject of "Goldstein's Heredofamilial Angiomatosis with Hemorrhages", with lantern demonstration, before the Hungarian Dermatologic Society at Budapest, the Professor Baron A. Korányi Medical Clinic, and the Professor Nekám Dermatologic Clinic of the Royal University, both at Budapest; before the German physicians of Carlsbad, Czecho-Slovakia; the American Medical Association, of Vienna; and also before the Professor Morawitz Medical Clinic, Leipzig University, Leipzig, Germany.

Dr. A. L. Anderson (Fellow), Springfield, Mo., on November 12, gave a paper before the Southwest Missouri Medical Society on "Metabolimetry as a Measure of Function in the Field of Clinical Medicine".

Dr. Chas. Hugh Neilson (Fellow), of St. Louis spoke on the same program on "Newer Theories of Nephritis"; and at the banquet of the Society responded to the toast, "Observation in Medical Education and Medical Practice".

Dr. Sidney Alexander Slater (Fellow), Worthington, Minn., addressed the Southwestern Minnesota Medical Society at their semiannual meeting, October 6, 1931, on his recent tour of European clinics.

On December 3, Dr. Slater addressed the Sioux Valley Medical Society at its annual meeting on the same subject.

The Eighty-Fifth Semi-Annual Meeting of the Southern California Medical Association was held in Hollywood, November 13 and 14, 1931, under the presidency of Dr. Fred B. Clarke (Fellow), Long Beach, Calif. Other Fellows of the College who held offices were:

Dr. William H. Barrow, San Diego, Calif.—First Vice President,

Dr. Carl R. Howson, Los Angeles, Calif.—Secretary-Treasurer.

At the above meeting, Dr. Ross Moore (Fellow), Los Angeles, delivered a paper on "A Concept of Toxic Activity—Therapeutic Application", and Dr. Samuel Ayres, Jr. (Fellow), Los Angeles, in conjunction with Dr. Nelson P. Anderson, delivered an address on "The Use of the Patch Test in the Diagnosis of Contact Dermatitis".

Dr. Clarence L. Andrews (Fellow), Atlantic City, N. J., was elected President of the Atlantic County (N. J.) Medical Society, December 11, 1931.

Dr. Arthur C. Morgan (Fellow), Philadelphia, addressed the annual meeting of the Sixth District Branch of the Medical Society of the State of New York, at Waverly, N. Y., September 22, 1931, on the subject "The Treatment of Acute Cardiac Tragedies".

Dr. Fred H. Voss (Fellow), Kingston, N. Y., has been elected a member of the New York Academy of Medicine.

On November 10, 1931, Dr. George A. Merrill (Fellow), Brooklyn, N. Y., addressed the Bay Ridge Medical Society on the subject "Allergy and its Common Manifestations".

Dr. Ray M. Balyeat (Fellow), Oklahoma City, Okla., Associate Professor of Medicine, University of Oklahoma Medical School, addressed The Medical and Surgical Association of the Southwest, at Phoenix, Arizona, December 4th on "History Taking and Etiology in Headaches Due to Specific Hypersensitiveness."

Dr. Balyeat recently has been elected President of the Oklahoma City Clinical Society.

At the Louisiana Follow-Up of the White House Conference on Child Health and Protection, November 12-14, at Baton Rouge, Dr. Ellen C. Potter (Fellow), Trenton, N. J., conducted a round table on the "Functions of Government in Public Welfare", and addressed the general assembly on the responsibility of government in that field.

In New Orleans, La., November 15-16, Dr. Potter delivered addresses before the Branch of the Medical Women's National

Association and before the City Officials and Private Social Agencies concerning the "Interdependence of Public and Private Social Work".

Dr. Potter addressed the Annual Convention of the New Jersey Organization for Public Health Nursing at Trenton, December 4, on "The Responsibility of Board Members". The same day, Dr. Potter also addressed the State Conference of Social Work of New Jersey on "Community Organization for Social Work".

The Second District Branch of the Medical Society of the State of New York held its Twenty-fifth Annual Meeting at the St. George Hotel, Brooklyn, November 19, 1931. Dr. Henry M. Moses (Fellow), Brooklyn, delivered an address on "Supportive Treatment of Pneumonia"; Dr. Albert F. R. Andresen (Fellow) and Dr. Simon R. Blatteis (Fellow), both of Brooklyn, delivered addresses on "Health Examination from the Standpoint of Gastro-enterology" and "Health Examination from the Standpoint of Internal Medicine", respectively.

Dr. Joseph H. Barach (Fellow), Pittsburgh, Pa., addressed the Westmoreland County Medical Society, November meeting at Mount Pleasant, Pa., on "Treatment of Pneumonia."

Dr. Robert A. Knox and Dr. George W. Ramsey (Fellows), Washington, Pa., presented a case of Niemann-Pick's disease before the Pittsburgh Pediatric Society, October 23. This case will be described in a paper which has been accepted for a future number of the ANNALS.

Dr. Fred M. Meixner (Fellow), Peoria, Ill., is the author of an article on "Chest Injuries as the Cause of Heart Lesions" in the December issue of the Illinois Medical Journal.

Dr. M. Murray Peshkin (Fellow), New York City, addressed the Hackensack Hospital Staff Association, Hackensack, N. J., Dec. 15, 1931, on "Allergy in Children".

OBITUARIES

DR. EDWARD TYLER EDGERLY

Dr. Edward Tyler Edgerly, Fellow of the College since March 10, 1925, died in Rochester, Minn., on November 15, 1931, following an operation for vesical calculus, at the age of sixty-seven years.

Doctor Edgerly was born in Ottumwa, Iowa, January 15, 1864. After attending the Ottumwa public schools he graduated from Phillips Exeter Academy and received his A.B. degree at Harvard University in 1885. He graduated in medicine from Northwestern University Medical School in 1889. In the competitive intern examination for Cook County Hospital, Chicago, he received the highest grade and became chief of the resident intern staff. After completion of his intern service, he was appointed an instructor in medicine and physical diagnosis of his Alma Mater.

In 1894, while on a tour of European medical centers accompanied by his father, Mr. J. W. Edgerly, the latter died suddenly in Paris, and this unfortunate occurrence changed the course of Doctor Edgerly's career. In the interest of the family fortunes, Doctor Edgerly made the willing sacrifice of a promising medical future in Chicago and devoted his services to the business interests of the family until 1908, when he returned to his old love and began the active practice of medicine in his home city. His fine training and high mental endowments

soon brought him into leadership among the physicians of his state.

In 1902 he was commissioned First Lieutenant in the Medical Reserve Corps, U. S. Army, and entered active service at the beginning of the World War. He served as Captain and Major, and Chief of the Medical Service of the Base Hospital at Camp Dodge during the period of the War. During Major Edgerly's service at Camp Dodge his conduct of the epidemics of meningitis and influenza received official commendation.

He was a member of the Wapello County and Iowa State Medical Societies, the Iowa Clinical Medical Society (past president), and the American Medical Association. He was an active member of the Iowa Tuberculosis and the Iowa Heart Associations.

Doctor Edgerly was married in 1891 to Miss Nettie Thurston of Chicago, who with a son, John T. Edgerly, and a daughter, Mrs. Nelson Rupe, and four grandchildren survive.

Since his affiliation with the College he has been an active worker in promoting its interests in every way and a faithful attendant at the annual meetings. During the Boston session in 1929 the members of his Harvard class (L.A. '85) arranged a dinner in his honor, and it was the writer's privilege to be present as a guest. Among those attending the dinner were Mr. Roland W. Boyden, eminent authority on international law and later successor to Justice Charles H. Hughes on the Court at the Hague; his brother, Mr. Wm. W. Boyden, a leading member of the Chicago Bar; Dr. Horace D. Arnold of Boston, a president of one of the leading life insurance companies; and others prominent in the professions and public affairs. Greetings were read from Dr. W. S. Thayer of Baltimore, and Associate Justice Edward T. Sanford of the U. S. Supreme Court. The late Dr. Lawrence Litchfield of Pittsburgh was also a member of this class.

Doctor Edgerly distinctly contributed to the progress of scientific medicine during his period, and the impression of his fine and genial personality, generous nature, and high professional ideals will linger with us while memory lasts.

(Furnished by Walter L. Bierring, M.D., F.A.C.P., Des Moines, Iowa.)

DR. JOHN FRANCIS WALLACE MEAGHER

Dr. John Francis Wallace Meagher (Fellow), Brooklyn, New York, died August 25, 1931, of injuries received when a concrete pillar supporting an observation platform on which he was standing collapsed. He was fifty-one years old and in the prime of health and vigor.

Dr. Meagher was born in Brooklyn, March 22, 1880. He was graduated from the Boys High School of Brooklyn and from the College of Physicians and Surgeons of Columbia University in 1901. He was an intern at St. Mary's Hospital, Brooklyn, from 1901 to 1902. In 1903 he made a special study of nervous and mental diseases at St. Lawrence State Hospital, Ogdensburg, New York, and later at the Manhattan State Hospital for the Insane at Ward's Island, New York. Thereafter he was associated in this work at the Bellevue Psychopathic Ward and in the Neurological Insti-

tute. During the World War he served in the rank of major. At first he was stationed at Camp Mills, Mineola, and later at Kelly Field, San Antonio. He was sent overseas and worked at Base Hospital 37, Dartford, Kent, and after that at Base Hospital 216 at Savenay, France. After the war was over he remained in the service in the capacity of psychiatrist at the Army Hospital at Plattsburg, New York, and was discharged in 1919.

At the time of his death, Dr. Meagher was Consultant Psychiatrist at Kings Park State Hospital and Neurologist at St. Mary's, Mary Immaculate, and Rockaway Beach Hospitals.

Dr. Meagher was a frequent contributor to medical literature and was associate editor of the Medico-Legal Journal and associate editor of the Journal of Urology and Cutaneous Diseases. His special interest, however, was in the legal and criminal aspects of mental abnormalities and diseases. For many years he had been a consultant alienist in the office of the District Attorney of Brooklyn, where his talents and abilities, along his special lines, were recognized as invaluable to the solution of their problems.

He was a member of the Pi Lambda Phi and the Chi Zeta Chi Fraternities. He was a member of the American Medical Association, New York Academy of Medicine, Neurological Society of New York, Neurological Society of Brooklyn, American Psychiatric Association, Medical Society of the County of Kings, and the Society of Medical Jurisprudence. He was also a member of the Crescent Athletic Club and the Cavalry Club of Brooklyn. He became a Fellow of the American College of Physicians in 1920.

(Furnished by Luther F. Warren, M.D., F.A.C.P., Governor of Eastern New York.)

DR. LAWRENCE EVANS CHAPMAN

Dr. Lawrence Evans Chapman (Internal Medicine) was born August 28, 1887, at Georgetown, Texas; received his M.D. degree from the University of Texas, School of Medicine, 1915; and became a member of the Galveston County Medical Society, the South Texas District Medical Society, State Medical Association of Texas, Southern Medical Association and American Medical Association. He was elected a Fellow of the American College of Physicians, March 4, 1928. At the time of his death Dr. Chapman was Associate Professor of Clinical Medicine at the University of Texas School of Medicine, and a visiting physician on the staff of the John Sealy Hospital. He died suddenly, October 21, of coronary thrombosis.

Dr. Chapman was a man of the highest principles, of the utmost devotion to the teaching and practice of Internal Medicine, and a most excellent type of gentleman. His death came as a great loss to the Medical Department of the University of Texas, with which he had been associated in various positions since 1915. He first became associated with the faculty as Instructor in Physiology, which position he held until 1917. After his training in Physiology, he became connected with the Chair of Practice of

Medicine where he continued until his death, passing through successive and rapid promotions from Instructor to the rank of Associate Professor.

(Furnished by C. T. Stone, M.D., F.A.C.P., Governor for Texas.)

DR. THOMPSON FRAZER

Dr. Thompson Frazer (Fellow), Newark, New Jersey, died suddenly October 9, 1931, of cerebral hemorrhage; aged fifty-four years.

Dr. Frazer was born in Buffalo, New York, September 10, 1877. He attended the Newark Academy of Newark, New Jersey, and later Princeton University, from which he received the degree of B.S. in 1897, and M.S. in 1904. He received his medical training at the College of Physicians and Surgeons at Columbia University, receiving his medical degree in 1901.

Dr. Frazer practised medicine in Asheville, North Carolina, for some time, later removing to Newark, New Jersey, where he established himself. Among his appointments was that of Visiting Physician, St. Barnabas Hospital. He was a member of his county and state societies, Southern Medical Association, The American Heart Association, American Climatological and Clinical Association, and a Fellow of the American Medical Association. He was elected an Associate of the American College of Physicians on March 13, 1925, and his credentials for Fellowship were accepted on December 30, 1926.